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SUBSTITUTE CHEMICAL PROGRAM

INITIAL SCIENTIFIC AND MINIECONOMIC REVIEW OF PARATHION

Midwest Research Institute

Prepared for

Environmental Protection Agency

January 1975

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This is the first of a series of in-depth scientific literature searches which review the safety and efficacy of substitute chemicals registered by EPA as possible replacements for cancelled or suspended pesticides or pesticides in litigation. The reviews are part of the Substitute Chemical Program, initiated under PL 93-135 (October 1973). This report on parathion, a pesticide substituting for DDT, reviews the substitute chemical's chemistry and analytical methods, its toxicity to laboratory and domestic animals and to humans. its effects on the environment, and its use patterns, costs and economic efficacy. The review is a compilation and summarization rather than an interpretation of published and unpublished scientific test and use data from various sources, including: journals, books, Federal and State agency reports, and manufacturer and university reports.

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SUBSTITUTE CHEMICAL PROGRAM

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OF
PARATHION

JANUARY 1975

U.S. ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF PESTICIDE PROGRAMS
CRITERIA AND EVALUATION DIVISION
WASHINGTON, D.C. 20460

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PREFACE

The Alternative (Substitute) Chemicals Program was initiated under Public Law 93-135 of October 24, 1973, to "provide research and testing of substitute chemicals." The legislative intent is to prevent using substitutes, which in essence are more deleterious to man and his environment, than a problem pesticide (one that has been suspended, cancelled, deregistered or in an "internal review" for suspected "unreasonable adverse effects to man or his environment"). The major objective of the program is to determine the suitability of substitute chemicals which now or in the future may act as replacements for those uses (major and minor) of pesticides that have been cancelled, suspended, or are in litigation or under internal review for potential unreasonable adverse effects on man and his environment.

The substitute chemical is reviewed for suitability considering all applicable scientific factors such as: chemistry, toxicology, pharmacology and environmental fate and movement; and socio-economic factors such as: use patterns and costs and benefits. EPA recognizes the fact that even though a compound is registered it still may not be a practical substitute for a particular use or uses of a problem pesticide. The utilitarian value of the "substitute" must be evaluated by reviewing its biolcgical and economic data. The reviews of substitute chemicals are carried out in two phases. Phase I conducts these reviews based on data bases readily accessible at the present time. An Initial Scientific Review and Mini-Economic Review are conducted simultaneously to determine if there is enough data to make a judgment with respect to the "safety and efficacy" of the substitute chemical. Phase II is only performed if the Phase I reviews identify certain questions of safety or lack of benefits. The Phase II reviews conduct in-depth studies of these questions of safety and cost/benefits and consider both present and projected future uses of the substitute chemicals.

The report summarizes rather than interprets scientific data reviewed during the course of the studies. Data is not correlated from different sources. Opinions are not given on contradictory findings.

This report contains the Phase I Initial Scientific and Mini-Economic Review of parathion (0,)-dimethyl 0-p-nitrophenyl phosphorothioate). Parathion was identified as a registered substitute chemical for certain cancelled and suspended uses of DDT. Where applicable, the review also identifies areas where technical data may be lacking so that appropriate studies may be initiated to develop desirable information.

The review covers all uses of parathion and is intended to be adaptable to future needs. Should parathion be identified as a substitute for a problem pesticide other than DDT, the review can be updated and made readily available for use. The data contained in this report was not intended to be complete in all areas. The review was coordinated by a team of EPA scientists in the Criteria and Evaluation Division of the Office of Pesticide Programs. The responsibility of the team leader was to provide guidance and direction and technically review information retrieved during the course of the study. The following EPA scientists were members of the review team: George Bagley (Chemistry), team leader; Jeff Conopask (Economics); E. David Thomas (Registered Uses); Ralph C. Wright (Registered Uses); William Burnam (Pharmacology and Toxicology); Elsie Kelley (Pharmacology and Toxicology); Howard Kerby (Fate and Significance in the Environment); Richard Tucker (Fate and Significance in the Environment).

Data research, abstracting and collection were primarily performed by Midwest Research Institute, Kansas City, Missouri (EPA Contract #68-01-2448). RvR Consultants, Shawnee Mission, Kansas, under a subcontract to Midwest Research, assisted in data collection. Monsanto, a manufacturer of parathion, made scientific recommendations and additions to this report. The recommendations of the following National Environmental Research Centers, EPA Office of Research and Development have also been incorporated: Southeast Environmental Research Laboratory, Athens, Georgia; Pesticide and Toxic Substances Effects Laboratory, Research Triangle Park, North Carolina.

PART I. SUMMARY

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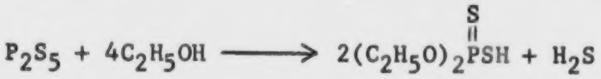
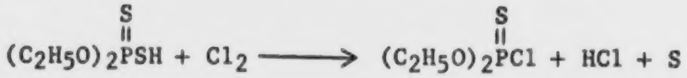
This section contains a summary of the "Initial Scientific and Minieconomic Review" conducted on parathion.

Production and Use

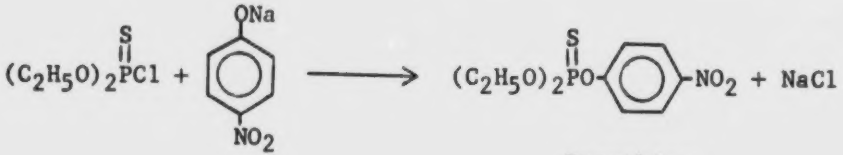
Parathion (O,O-diethyl O-p-nitrophenyl phosphorothioate) is manufactured by a synthesis processing involving three reactions:



$$P_2S_5 + 4C_2H_5OH \longrightarrow 2(C_2H_5O)_2\overset{\overset{\displaystyle S}{\parallel}}{P}SH + H_2S \qquad (1)$$



$$(C_2H_5O)_2\overset{\overset{\displaystyle S}{\parallel}}{P}SH + Cl_2 \longrightarrow (C_2H_5O)_2\overset{\overset{\displaystyle S}{\parallel}}{P}Cl + HCl + S \qquad (2)$$



Parathion

(3)

The chemical properties of parathion are similar to those of its methyl analog (methyl parathion). Parathion is oxidized enzymatically to paraoxon (O,O-diethyl O-p-nitrophenyl phosphate), a reaction intimately associated with parathion's biological effects. The oxidation of parathion to paraoxon can also be effected by chemical action of atmospheric oxygen; atmospheric oxidation is accelerated by ultraviolet radiation.

Hydrolysis of parathion virtually destroys all insecticidal activity. Fifty percent hydrolysis of parathion is achieved in 2.7 hr in aqueous solutions at pH 9 and 70°C; in 17 to 20 hr at pH 1 and 70°C; and in 690 days at pH 1 to 5 and 20°C. Fifty percent hydrolysis of paraoxon requires 9.2 days in aqueous solution at pH 10 and 25°C.

Parathion is commercially available in a large number of registered formulations and in many physical forms, including emulsifiable concentrates, wettable powders, dusts, granules, and aerosol formulations containing only parathion or parathion in combination with other active ingredients.

Parathion has a very broad spectrum of effectiveness against insects and mites. Tolerances for parathion residues have been established on about 100 raw agricultural commodities.

The estimated domestic production of parathion in 1972 is 14 million pounds. About 10 million pounds (as active ingredient) were used in the United States in 1972. A review of available data on farm use of parathion for the period 1966 to 1972 indicates an upward trend of 2%/year in the usage of parathion in agriculture. Two of the previous domestic producers of parathion, however, have discontinued production of this insecticide since 1972. There is no assessment possible on the ability of industry to increase parathion supply.

Essentially all domestic usage of this pesticide is agricultural. Parathion is registered and recommended for use on a large number of domestic crops, including fruit, nut, vegetable, and field crops.

Domestic crops on which parathion was used in 1972, listed in decreasing order of the quantity used, are: cotton, sorghum and small grains; deciduous fruit and nut crops; vegetable crops; corn; citrus fruits; tobacco; and numerous other crops, each of which account for a relatively small portion of the total usage.

The 1972 domestic use pattern, by geographical region and in decreasing order of use, is estimated to be: West South Central (Oklahoma and Texas); Southwest (California, Arizona, Hawaii, New Mexico, and Nevada); Southeast; West North Central; East South Central; Northwest; and Northeast. Slightly more than 10% of all parathion used in the U.S. in 1972 was applied in California on almost 100 different crops.

Pharmacology and Toxicology

Parathion is a highly toxic pesticide, as evidenced by studies with laboratory and domestic animals, fish, other aquatic life, avian species, wildlife and humans.

The literature on clinical cases of acute parathion poisoning in man is extensive. In nearly all instances, however, the amount ingested, inhaled, or adsorbed was unknown. An oral lethal dose is estimated to be 1-to-2 mg/kg of body weight; the largest nonfatal dose reported is 6.4 mg/kg.

A 16% inhibition of RBC cholinesterase has been produced in man at a dosage level of 0.1 mg/kg/day over a 42-day period, while the ingestion of 7.5 mg/day of parathion for 9 days produced a reduction of plasma cholinesterase of 27 to 32%.

The major cholinergic effects produced by parathion poisoning are weakness, nausea, vomiting, excessive sweating, headache, excessive salivation, difficulty in walking, miosis, and muscle fasciculations. Electroencephalographic (EEG) changes resemble those seen in epileptics.

There have been a few controlled investigations of the dermal effect of parathion on humans. No certain conclusions have been drawn from skin tests related to rates of absorption. Paraoxon, the oxidation product of parathion, however, does not appear to be hydrolyzed in the skin. In the controlled studies reported, the exposure of hand, forearm or whole body (nude) to relatively high concentrations of parathion had little adverse effect. Exposure to parathion vapors for 3 hr did not significantly reduce plasma cholinesterase activity.

The hazard from inhalation is said to be three times greater than from oral ingestion. Unfortunately, the test results that were reviewed do not report the actual concentration of parathion in the air that was being breathed. RBC cholinesterase and plasma cholinesterase depressions of 98 and 83%, respectively, were reported for subjects exposed to fumes from parathion.

The acute toxicity of parathion to male rats is presented below:

Route of entry	Measurement	Value
Oral	LD_{50} (mg/kg)	7.6
Intraperitoneal	LD_{50} (mg/kg)	3.6-7.0
Dermal	LD_{50} (mg/kg)	21.0
Inhalation	LC_{50} (1 hr) (mg/ℓ)	0.115
	LC_{50} (4 hr) (mg/ℓ)	0.0315

The collective data indicate that parathion is highly toxic to all species tested thus far by all routes of administration. There is usually a lesser degree of toxicity produced by dermal exposure than by other routes. The vehicle is very important in dermal toxicity tests. Episodes of acute toxicity are more likely to occur in nature than are chronic poisonings. The pathology of chronic toxicity is similar to that found in acute episodes. The young are more susceptible than the mature organisms. In rats, males appear to be less susceptible than females. The symptoms and mechanisms of physiological action in all species are essentially the same. Cholinesterase depression is a sensitive clinical measurement. Pathological damage appears to be minimal even in chronic exposures.

There is little available literature regarding parathion's effect on reproduction. Most of the reported studies were conducted on wild avian species (ducks, pheasants, and partridges). Findings indicated that 5-to-10 ppm of parathion had little effect, if any, on egg production, fertility or progeny growth. There was no indication that parathion residues occur in the egg.

There were only two studies found on the mammalian teratogenesis of parathion. Although they showed that parathion caused a reduction in the number of fetuses and an increase in the number of resorptions, they provided little evidence that actual malformation of the fetus occurs as a result of parathion exposure. The data suggests that parathion does not produce specific teratogenic effects.

A large number of papers have been published regarding the effect of parathion on the normal development of avian embryos. Various deformities have been produced when eggs are immersed in parathion or the pesticide is introduced into the yolk sac. Investigators have reported lordosis of the neck, celosomia, retardation of the axial skeleton, disorganized vertebral musculature, light edema, achondroplasia and abnormal leg position. Parathion reportedly acts as an antimitotic substance that markedly effects cell division. Generally, the dosage of parathion was relatively high before deformation of the embryo occurred.

The available data concerning the effect of parathion on crustaceans is meager. The normal length of oyster larvae, however, is reported to be greatly depressed by 1-ppm parathion in water.

Treatment of tissue cultures with parathion does not permit prediction of the activity that would be produced in the intact organism. In a study with HeLa cells (a human malignant cell line) paraoxon was less toxic than parathion, and nitrophenol was as toxic as parathion. Furthermore, relatively high amounts of parathion have to be used in tissue culture to produce a definitive response. There is little difference between malignant and nonmalignant cell responses to parathion.

There is practically no available literature on the induction of chromosomal abnormalities by parathion. However, abnormalities have been induced by parathion in mitotic chromosomes of the guinea pig.

No references were found on the oncogenic effect of parathion. Parathion had no clear effect on chemically induced tumors.

The following major points can be stated regarding the metabolism of parathion:

1. Parathion is readily adsorbed through the skin, from the stomach and the lung.
2. It becomes widely distributed throughout the body but does not accumulate at any site and causes no effect other than the irreversible binding of paraoxon to cholinesterase.
3. The major metabolites of parathion that are excreted are aminoparathion, p-nitrophenol, diethylphosphorothioic acid, ethylphosphoric acid and sulfate.
4. The biotransformation of parathion involves two distinct phenomena: activation and detoxification.
5. Parathion is enzymatically oxidized to paraoxon by the mixed function oxidase system.
6. Microsomal oxidation of parathion requires $NADPH_2$, oxygen, potassium chloride and magnesium ion for maximum activity.

Food Tolerances and Acceptable Intake

Tolerances for parathion have been established for about 100 raw agricultural commodities in the United States; all but one are in the 0.1 to 1.0 ppm range. The tolerances for parathion and methyl parathion are the same. When both chemicals are present, the tolerances apply to the combined total of methyl parathion and parathion.

The acceptable daily intake (ADI) for parathion has been set at 0.005 mg/kg body weight.

Environmental Effects

Parathion is toxic to fish and wildlife; product labels carry a warning that wildlife in treated areas may be killed. A brief summary of the acute toxicity of parathion to fish, anthropods and avian species is as follows:

LC_{50} for Fish (ppm)

Bluegill	24 hr	0.141
Bluegill	48 hr	0.047
Golden shiner	24 hr	0.931
Greenfish	24 hr	0.155
Mummichog	48 hr	0.15
Stripped mullet	96 hr	0.125
Tilapia	96 hr	0.375

LC_{50} for Anthropods (ppm)

Amphipod (*Gammarus lacustris*)	24 hr	0.012
Amphipod (*Gammarus lacustris*)	48 hr	0.006
Stonefly (*Claassenia sabulosa*)	24 hr	0.0088
Stonefly (*Pteronarcella badia*)	24 hr	0.008
Stonefly (*Pteronarcys californica*)	24 hr	0.028
Stonefly (*Pteronarcys californica*)	48 hr	0.011
Stonefly (*Pteronarcys* sp.)	48 hr	0.011
Waterflea (*Daphnia pulex*)	48 hr	0.0004
Waterflea (*Daphnia* sp.)	48 hr	0.00076

Oral LD_{50} for Avian Species (mg/kg)

Chukar partridge	24.0
House sparrow	1.3-3.4
Japanese quail	6.0
Mallard duck	1.0-2.1
Pheasant	12.4
Pigeon	2.5
Quelea	1.8
Red-winged blackbird	2.4
Starling	5.6

Parathion is universally recognized as being highly toxic to bees. Studies of the toxicity of parathion to bees (Apis mellifera) by direct topical application and by exposure to parathion containing solutions, vapors and surface deposits have shown this pesticide to be highly toxic by all routes tested.

The reports reviewed during this study indicate that parathion would permit survival of beneficial insects in some predator-parasite systems. For example, the LD_{50} of parathion to the English grain aphid (Macrosiphum aenae) is 1.8 µg/g, while the LD_{50} for two of the aphids most numerous ubiquitous predators (Hippodamia convergens and Nabis americoferus) are 40 and 21 µg/g, respectively. Such a favorable relationship, however, does not exist in all systems.

The available data also indicate that parathion is extremely toxic to aquatic fauna, but relatively nontoxic to the lower aquatic flora. A number of aquatic microorganisms degrade parathion. Many of these organisms also preferentially sorb and thus accumulate parathion from aqueous media. This sorption process may be independent of life processes; sorption rates of live and dead organisms showed little differences.

Only limited data was found on the presence of parathion in the air. One study reported on the parathion content of air inside and outside the homes of workers occupationally exposed to this insecticide. The results indicated that parathion was present in the atmosphere, thereby exposing the total population of the area. The presence of parathion residues in the air raises important questions, including the origin of such residues, their relationships to parathion use and handling patterns in the area, their persistence and transport patterns in air, and their significance to human and environmental health.

The persistence of parathion in the natural aquatic environment is apparently markedly affected by microbial activity. One study indicated that without microbial activity, parathion would remain in the environment for several months; in biologically active (aerobic or anaerobic) environments, it would be degraded in a matter of weeks.

Other investigators have concluded that it is unlikely that water in deeper soil strata could become contaminated with insecticidal residues from the upper agricultural soil layers, or that parathion could contaminate underground water supplies beneath any of these soils by leaching under normal rainfall conditions.

Laboratory, field, and monitoring data on the residues and fate of parathion in the soil show that the persistence of this chemical varies considerably, depending upon a number of factors. Parathion residues in the soil degrade by chemical hydrolysis as well as by microbial action. The half-life of parathion in soil has been shown to be as short as 1.5 week. In the case of heavy soil contamination, parathion residues have been shown to persist for at least 16 years.

Parathion is strongly adsorbed by organic matter. Once adsorbed the chemical becomes unavailable for insecticidal action, and appears to be protected to a degree against degradation. No data is available on the fate of such adsorbed parathion residues; it is not known whether they eventually degrade, or if and under what conditions they might become reactivated by desorption. No information appears to be available on the fate of the initial degradation products, especially p-nitrophenol and aminoparathion, or on the effects of these degradation products on organisms other than mammals and insects.

The two most important environmental transport mechanisms for parathion appear to be volatilization and surface runoff while absorbed on solids.

No data were found on the biomagnification of parathion.

Specific Hazards of Use

The only major hazards associated with the registered uses of parathion that have been documented by this review are its acute toxic hazard to man and, to a lesser degree, its acute toxic hazard to many of the other higher organisms. Data were not found that documented the association of this hazard with any specific use of parathion.

Limitations in Available Scientific Data

The review of the scientific literature was based on available sources, given limitations of time and resources. Data was not found in a number of pertinent areas:

1. The mechanism of tolerance to chronic parathion administration.
2. Chronic inhalation toxicity of parathion, based on cholinesterase inhibition and on unmistakable signs of toxicity.
3. The fate of parathion residues adsorbed on organic matter or other soil colloids.
4. The fate and effect in the environment of the major degradation products of parathion, especially p-nitrophenol and aminoparathion.
5. The persistence and fate of parathion residues in water, especially under field conditions.
6. The origin, presence, persistence and significance of parathion residue in air.
7. Parathion metabolism and residues in and on nontarget plants, and regarding the effects of these residues on wildlife feeding on such plants.
8. Effects on ecosystems.

Efficacy and Cost Effectiveness

Parathion is registered for a wide variety of crops and pests; yield changes and efficacy data have been reported for numerous crop-pest combinations. However, because of inherent limitations in crop-test data, only order-of-magnitude estimates of economic benefits can be made.

Parathion was reported to be effective when used on cotton to control the bollworm and boll weevil, but was much less effective for control of the tobacco budworm. The budworm is more resistant to parathion than to methyl parathion. The results of one yield test showed economic gains by the use of parathion on cotton of $93.98/acre.

Effective control of the sorghum midge was achieved with parathion. However, economic effects ranged from a loss of $10.00 to a gain of $5.00 per acre. Rate and timing of application were important to the effect on sorghum yield.

Parathion also controls the greenbug on sorghum. Reported yield changes were variable, ranging from a loss of 458 pounds to a gain of 1,083 pounds per acre; economic benefits ranged from a loss of $12.80 to a gain of $23.80/acre. Some phytotoxicity was observed, however, when high rates of parathion were used, and thus adversely affected yields from some varieties of sorghum.

Greenbug control on wheat ranged from 88% to 92%; economic benefits ranged from $5.70 to $17.00/acre from the use of parathion.

In two out of three tests conducted in Virginia, parathion greatly reduced the amount of damage to peanuts caused by the southern corn rootworm. A control was not used in the third test, and there was no explanation for not using it. Economic benefits for the one test reporting yields amounted to $26.50/acre.

One application of parathion effectively controlled the European corn borer and Western corn rootworm, with benefits ranging from $5.40 to $143.30/acre.

Application of parathion to control the Pacific wireworm on potatoes resulted in reductions of 78% to 95% in the amount of culls. Economic benefits varied from a loss of $103.00 to a gain of $108.70/acre.

Economic benefits for control of the potato aphid amounted to $87.90/acre. For the potato leafhopper, benefits were $504.50/acre.

Parathion applied to lima beans at the time of first bloom resulted in good control of Lygus hesperus and increased yields significantly. Economic benefits for one test were $236.70/acre.

The economic benefit for control of the pea aphid in one test was $67.50/acre.

Two applications of parathion appeared to provide satisfactory control of the tarnished plant bug on strawberries. The economic benefits ranged from $527.70 to $905.70/acre.

Cost effectiveness on a limited statistical basis was demonstrated for four major crops including three major cotton pests, two sorghum pests, one wheat pest, and two corn pests. Additional data were presented on three potato pests and three vegetable/fruit pests.

PART II. INITIAL SCIENTIFIC REVIEW

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This subsection contains a review of available data on parathion's chemistry and presence in foods. Eight subject areas have been examined:

1. Synthesis and production technology.
2. Physical properties of parathion.
3. Composition and formulation.
4. Chemical properties, degradation reactions and decomposition processes.
5. Occurrence of residues in food and feed commodities.
6. Acceptable daily intake.
7. Tolerances.

Synthesis and Production Technology

The chemical processes used for parathion and methyl parathion manufacture are almost identical. The only difference is in the alcohol (methanol or ethanol) used in the initial reaction (with phosphorus pentasulfide). The equipment for producing these compounds is identical. In most cases the same processing equipment is used for the manufacture of both products.

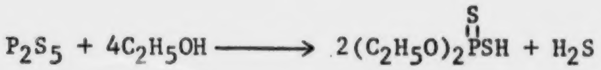
The following is a list of known parathion manufacturers and production capacity and levels.

Manufacturer and plant location	Plant capacity a/ (million lb/year)	Estimated 1972 production of parathion (million lb)
Monsanto Company Anniston, Alabama	50	10
Stauffer Chemical Company Mount Pleasant, Tennessee	30	3
Kerr-McGee Chemical Corporation		
Hamilton, Mississippi	9	None
Los Angeles, California	3	1
Velsicol Chemical Corporation Bayport, Texas	10	None
Total	102	14

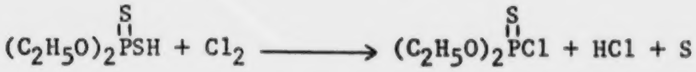
Source: MRI estimates.

a/ Plant capacity is stated for methyl parathion plus (ethyl) parathion. Stauffer and Kerr-McGee have discontinued parathion production since 1972.

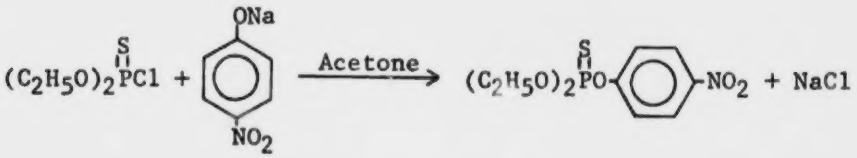
Three reactions are involved in the synthesis of parathion:



$$P_2S_5 + 4C_2H_5OH \longrightarrow 2(C_2H_5O)_2\overset{\overset{S}{\|}}{P}SH + H_2S \qquad (1)$$



$$(C_2H_5O)_2\overset{\overset{S}{\|}}{P}SH + Cl_2 \longrightarrow (C_2H_5O)_2\overset{\overset{S}{\|}}{P}Cl + HCl + S \qquad (2)$$



(3)

In the first two reactions, the intermediate diethyl phosphorothionochloridate is produced. This intermediate may be produced on site or elsewhere, or can be purchased from another manufacturer.

One of the most detailed descriptions of methyl parathion manufacture is a 1953 U.S. patent. Although this patent discusses methyl parathion manufacture, the process description is applicable to parathion synthesis. In the patent, Schrader (1953)[1/] describes the reaction 3 procedure for the manufacture of methyl parathion. According to Schrader's procedure, this exothermic reaction is performed at 80 to 100°C. However, according to a patent by Dvornikoff et al. (1953),[2/] it is preferred to operate at a much lower temperature of from -10 to +10°C. The reaction is performed at atmospheric pressure and requires a reaction time of 5 hr at 80 to 95°C but about 18 hr if performed at 0°C.

The reaction is conducted in the liquid phase. One of various organic solvents may be present. An alcoholic medium is specified by Dvornikoff et al. (1953). However, an inert solvent such as benzene or chlorobenzene is preferred by Schrader (1953). Monsanto employs acetone as solvent.

Copper powder may be used as a catalyst or the reaction may simply be conducted in a copper reaction vessel, thereby shortening reaction time appreciably. A small amount of potassium bromide can be used as an effective co-catalyst. Aliphatic amine catalysts were cited for use in this reaction by Toy et al. (1949).[3/] At least 0.25% of such materials as triethylamine, tributylamine, N-ethyl morpholine and hexamethylene tetramine may be used.

The reaction is carried out in a stirred, jacketed vessel. Although the reaction is conducted at essentially atmospheric pressure, closed vessels must be used because by-product gases are both toxic and odorous.

The yield of methyl parathion is 90% or higher. The reaction product mixture may be pumped through a precoated filter to remove gummy impurities. The filtrate may then be separated into aqueous and oily layers. The lower oily layer may then be washed with a dilute sodium carbonate solution, then with water; it may then be steam distilled to remove by-product trimethyl thiophosphate. After cooling and settling,

---

[1/] Schrader, G. (to Farbenfabriken Bayer), U.S. Patent No. 2,624,745 (6 January 1953).

[2/] Dvornikoff, M. N., et al. (to Monsanto Chemical Company), U.S. Patent No. 2,663,721 (22 December 1953).

[3/] Toy, A. D. F., et al. (to Victor Chemical Works), U.S. Patent No. 2,471,464 (31 May 1949).

the organic layer can be dried by heating under vacuum to give the product.

Monsanto produces parathion by a batch process in equipment dedicated to methyl and ethyl parathion production. Two of the raw materials are produced on site and the rest are received by rail. (See Table 1.) A production schematic is shown in Figure 1.

Stauffer's method of producing parathion is believed to be similar to Monsanto's.

No information is available concerning production methods at the Kerr-McGee methyl parathion plant. The plant at Hamilton does not produce either P_2S_5 or the p-nitrophenol.

Physical Properties of Parathion

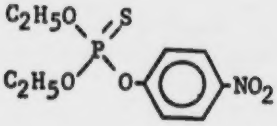
Chemical Name: O,O-Diethyl O-p-nitrophenyl phosphorothioate

Common Name: Parathion

Trade Names: Alkron, Alleron, Bladan, Corothion, E-605, Ethyl Parathion, Ethlon, Folidol E-605, Niran, Orthophos, Panthion, Paramar, Parathene, Parawet, Phoskil, Rhodiatrox, Soprathion, Stathion, Thiophos

Pesticide Class: Broad-spectrum nonsystemic insecticide; organophosphate

Structural Formula:



Empirical Formula: $C_{10}H_{14}O_5NPS$

Molecular Weight: 291.27

Analysis: C, 41.23%; H, 4.84%; O, 27.47%; N, 4.81%; P, 10.64%; S, 11.01%

Physical State: Pure compound is a yellow liquid. Technical material is an amber to dark brown liquid, with a garlic-like odor.

Table 1. RAW MATERIALS AND BY-PRODUCTS IN THE MANUFACTURE OF PARATHION

Raw Materials

	Material	Received from	Received by	Storage	Remarks
1.	P_2S_5	On-site	Tote bins	Tote bins	
2.	Cl_2	Louisiana	Rail, tanks	Tank cars	Vented to production system
3.	C_2H_5OH	Louisiana	Rail, tanks		
4.	$NaOC_6H_4NO_2$	On-site		Tank	
5.	Acetone	Southwest	Rail, tanks		
6.	Soda ash	East; middle-west	Rail, tanks	Bulk	For waste disposal

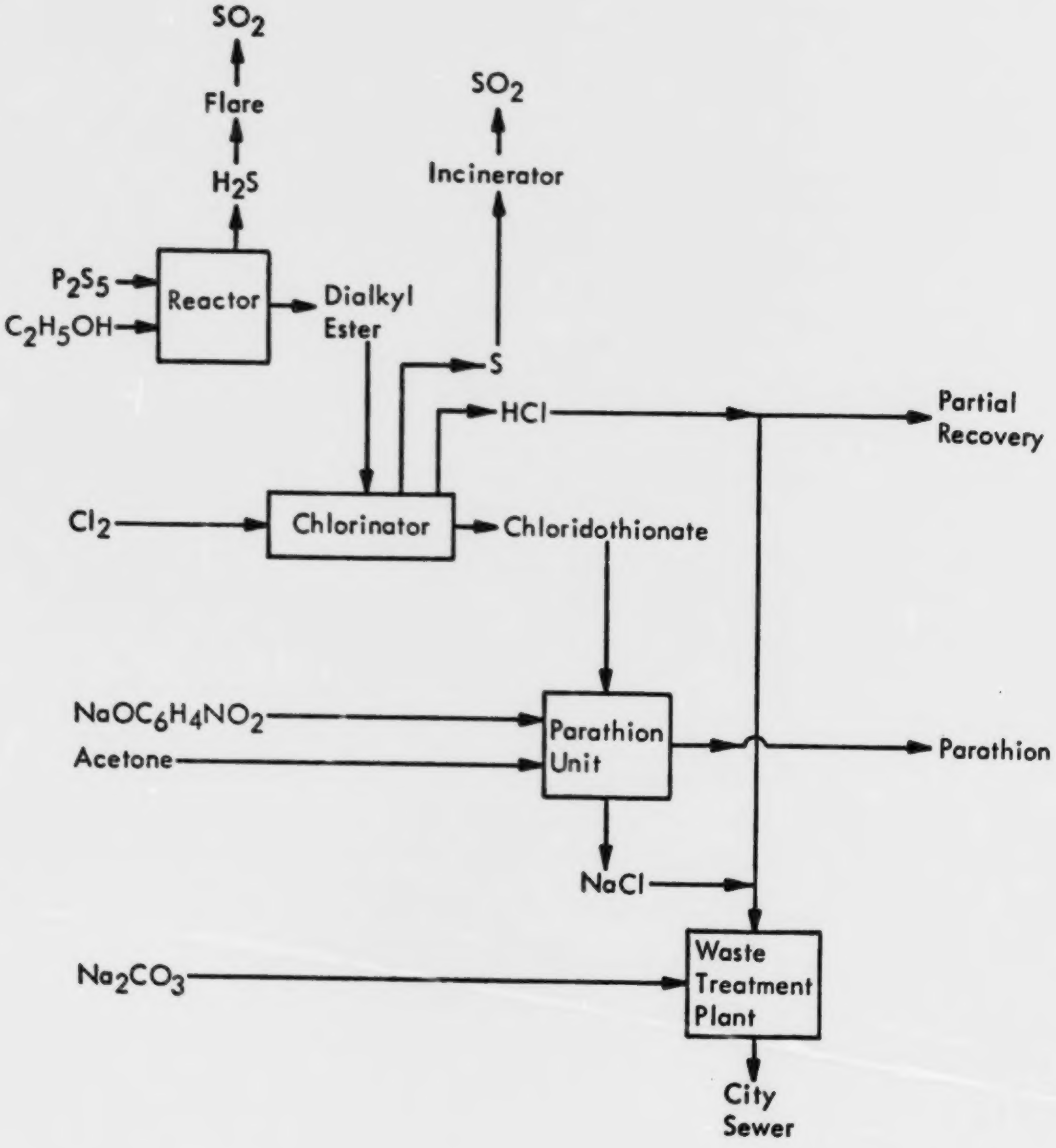
Reaction By-Products

	Material	Form	Amount produced (lb/lb AI)	Disposition	Remarks
1.	H_2S	Gas	0.06 calcd.	Flared	SO_2 air pollutant
2.	HCl	Gas	0.12 calcd.	Most recycled	Some to liquid waste
3.	S		0.11 calcd.	Incinerate	SO_2, some H_3PO_4?
4.	NaCl		0.20 calcd.	Biol. waste treatment	Discharged to city sewer

Other Process Wastes and Losses

Material	Form	Amount produced (lb/lb AI)	Disposition	Remarks
Active ingredient	Aqueous		Liquid waste treatment	< 1 ppm to city sewer
Solvents			Burned	
Other: Organophosphates	Gas liquid		Liquid waste treatment	
p-nitrophenol			Liquid waste treatment	

Source: Lawless, E. W., and T. L. Ferguson of Midwest Research Institute, and R. von Rümker of RvR Consultants, The Pollution Potential in Pesticide Manufacturing, for Environmental Protection Agency, Contract No. 68-01-0142 (January 1972).


SO_2
Flare
H_2S
SO_2
Incinerator
P_2S_5
C_2H_5OH
Reactor
Dialkyl Ester
S
HCl
Partial Recovery
Cl_2
Chlorinator
Chloridothionate
$NaOC_6H_4NO_2$
Acetone
Parathion Unit
Parathion
NaCl
Na_2CO_3
Waste Treatment Plant
City Sewer

Source: Lawless et al., op. cit. (1972).

Figure 1. Production schematic for parathion.

Specific Gravity: 1.26 at 20/20°C

Density: 10.5 lb/gal at 20°C

Melting Point: 6°C

Boiling Point: Thermally unstable and cannot be heated to normal boiling point, which is calculated to be 375°C. Various other boiling points are as follows:

Vapor pressure (mm Hg)	Temperature (°C)
0.57×10^{-5}	20
0.05	113
0.6	157-162

Bright et al. (1950)[1] determined the following vapor pressures:

3.78×10^{-5}	20
2.08×10^{-4}	40
9.31×10^{-4}	60
3.52×10^{-3}	80
1.15×10^{-2}	100

Refractive Index: $n_D^{25} = 1.5370$

Surface Tension: 39.2 dynes/cm at 25°C

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[1] Bright, N. F. H., J. C. Cuthill, and H. H. Woodbury, "The Vapor Pressure of Parathion and Related Compounds," *J. Sci. Food and Agr.*, 1:344-348 (November 1950).

Viscosity: 15.30 cp at 25°C

Solubility: Water at 25°C--24 ppm
Miscible with common organic solvents such as acetone, benzene, carbon tetrachloride, chloroform, ethanol, ether, toluene and xylene; slightly soluble in heptane and petroleum oils; slightly soluble in lipids and fats.

Flash Point (Tag Open Cup): 120°C

Volatility: 0.09 mg/m^3

Analytical Methods

This subsection reviews parathion analytical methods and the most significant of many primary information sources on the methods. The following information sources are described: (1) *The Pesticide Analytical Manual* (PAM), vols, I, II[1], (2) *Official Methods of Analysis of the Association of Official Analytical Chemists*[2], (3) *Analytical Methods for Pesticides and Plant Growth Regulators*[3].

The Pesticide Analytical Manual - *The Pesticide Analytical Manual* (PAM), published by the Food and Drug Administration, synthesizes procedures and methods used by FDA laboratories to examine food samples for the presence of pesticide residues.

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[1] U.S. Department of Health, Education, and Welfare, Food and Drug Administration, *Pesticide Analytical Manual*, 2 vols. (1971).

[2] Association of Official Analytical Chemists, *Official Methods of Analysis of the Association of Official Analytical Chemists*, 11th ed., Washington, D.C. (1970).

[3] Zweig, G., and J. Sherma, *Analytical Methods for Pesticides and Plant Growth Regulators, Vol. VI: Gas Chromatographic Analysis*, Academic Press, New York (1972).

The PAM is published in two volumes. Volume I contains procedures for multi-residue methods (for smaples of unknown history which may contain more than one pesticide). Volume II contains analytical methods used for specific pesticide residues and for specific foods.

Official Methods of Analysis of the Association of Official Analytical Chemists - The Association of Official Analytical Chemists (AOAC) publishes an authoritative methods manual about every 5 years. The manual is designed to provide both research and regulatory chemists with reliable methods of analysis. The reliability of the methods must be demonstrated by a published study showing the reproducibility of the method by professional analysts.

When an AOAC method is adopted for the first time it is published as "Official First Action." This designation serves notice that final adoption is pending, and permits an opportunity for any further study that may be deemed appropriate.

Methods that have performed successfully for at least 1 year are raised to the status of "Official Final Action."

A few methods are adopted as "Procedures." Such methods are generally sorting or screening methods or well-established types of examinations, or auxiliary operations, such as sampling or preparation of a sample, which may not have been subjected to collaborative study.

Analytical Methods for Pesticides and Plant Growth Regulators, Volume VI, Gas Chromatographic Analysis - Chapter 6 of this text consists of an extensive and detailed review of specific and multi-residue analytical methods for organophosphate pesticides. This reference provides important information not available in AOAC's "Methods of Analysis" or the PAM such as: (1) a comparison of nine procedures for extracting phosphorus insecticides and their metabolites from field-treated crops, (2) a review of procedures for extracting organophosphate pesticides from water samples, (3) a review of insecticide recoveries from vegetables, (4) a review of various clean-up procedures, (5) a description of various detectors, (6) extensive data comparing the relative retention times of various pesticides on various column materials, and (7) a review of the sensitivity of various gas chromatographic systems.

Multi-Residue Methods

Multi-residue methods for parathion are described in the AOAC methods manual and PAM, Volume I. Zweig and Sherma have compiled a detailed review of gas chromatographic residue analyses.

AOAC Methods - One of the AOAC methods,[1] a general method for "chlorinated and phosphated pesticides," is an "Official First Action" and applies only to apples and lettuce. A second AOAC multi-residue method[2] applies only to "phosphated pesticides" (in kale, endive, carrots, lettuce, apples, potatoes, and strawberries). This second method is also an "Official First Action" and involved a sweep codistillation cleanup for the organophosphate residues. (The cleanup is not adequate for electron capture detectors; KCl thermionic detectors must be employed.) Also described in AOAC methods manual[3] is a single sweep oscillographic-polarographic confirmation method.

The following AOAC multi-residue method is used for chlorinated and phosphated pesticides: A thoroughly mixed sample is extracted with acetonitrile. Aliquots of the acetonitrile solution are diluted with water and the pesticide residues are extracted into petroleum ether. The residues are purified by chromatography on a Florisil column and are eluted from the column with mixtures of petroleum and ethyl ethers. The first eluate, (6% ethyl ether in petroleum ether) contains some chlorinated pesticides and some phosphated pesticides. However, methyl parathion and parathion (and diazinon) are obtained in a second eluate (15% ethyl ether in petroleum ether). A third eluate (50% ethyl ether in petroleum ether) contains malathion. The eluates are concentrated, and the residues are determined by gas chromatography and identified by combinations of gas, thin-layer, or paper chromatography.

PAM Procedures - The PAM multi-residue methods (PAM, 1971) apply to the wide variety of foods tested by the FDA. However, the multi-residue methods are not capable of detecting and measuring all pesticides. Analytical schemes used in the detection of parathion are shown in Figures 1 and 2. The various parts of the schemes shown in Figures 1 and 2 are outlined in detail in the PAM. (The numbers refer to the chemical numbering system of PAM; the chapter numbers also refer to PAM.)

Parathion is more than 80% recovered in the 15% ethyl ether in petroleum ether fraction from the Florisil column. Over 80% recovery is achieved from both fatty and nonfatty foods.

Relative retention times of parathion are presented below for various column packings; the corresponding response for various detectors is also indicated.

---

[1] AOAC, op. cit., p. 475 (1970).
[2] AOAC, op. cit., p. 484 (1970).
[3] AOAc, op. cit., p. 487 (1970).

Electron Capture Detector

Column packing	Retention time relative to aldrin (ratio)	Response (ng for 1/2 FSD* at 1 x 10^{-9} AFS**)
10% DC 200 on Gas-Chrom Q (or Anakrom Q)	0.98	15
15% QF-1, 10% DC 200 on Gas-Chrom Q	1.88	5

Sulfur Detector

Column packing	Retention time relative to sulphenone (ratio)	Response (µg for 112 FSD* 64 ohs)
10% DC 200 on Gas-Chrom Q	0.78	1.5
15% QF-1, 10% DC 200 on Gas-Chrom Q	0.71	1.5

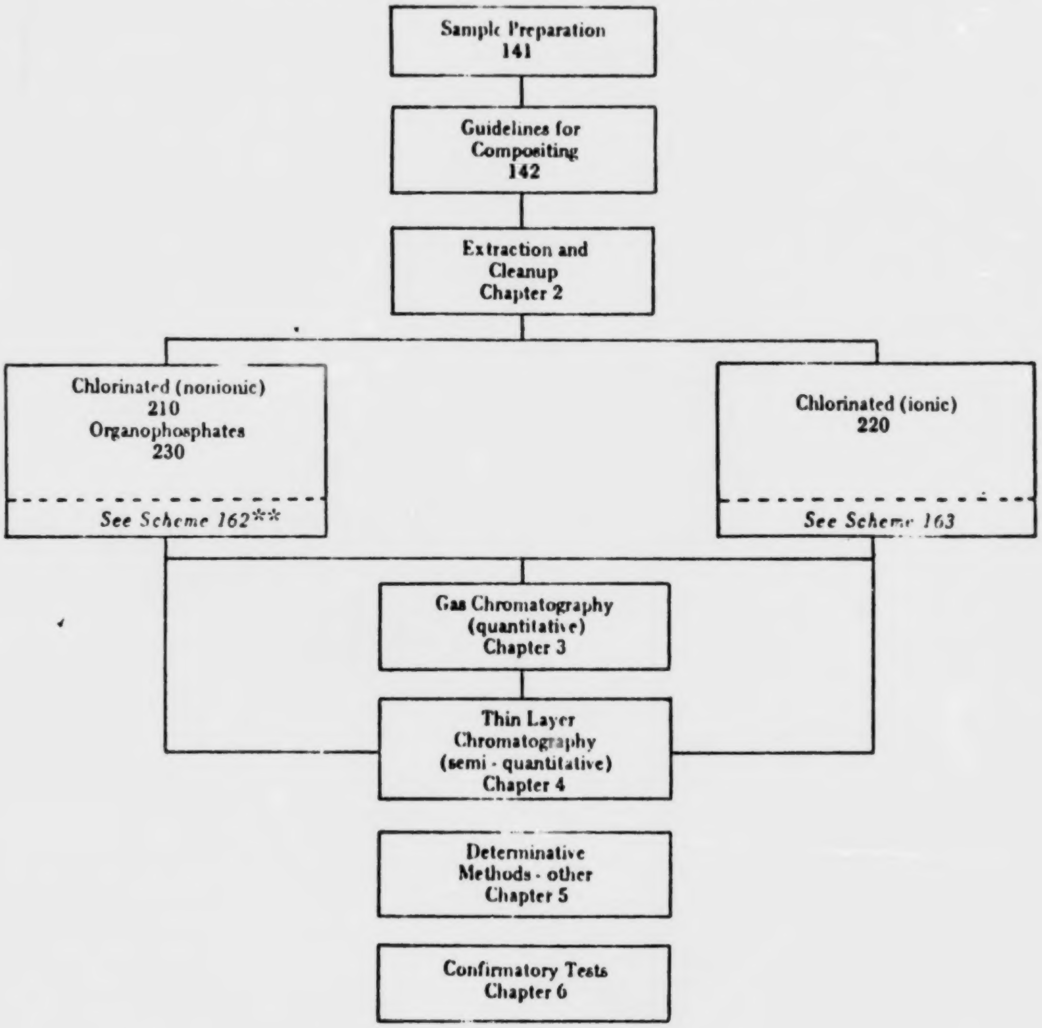
---

* FSD = Full scale deflection.

** AFS = Amps, full scale.

Figure 2

GENERAL SCHEME FOR MULTIPLE RESIDUES*


Sample Preparation
141
Guidelines for Compositing
142
Extraction and Cleanup
Chapter 2
Chlorinated (nonionic)
210
Organophosphates
230
See Scheme 162**
Chlorinated (ionic)
220
See Scheme 163
Gas Chromatography (quantitative)
Chapter 3
Thin Layer Chromatography (semi - quantitative)
Chapter 4
Determinative Methods - other
Chapter 5
Confirmatory Tests
Chapter 6

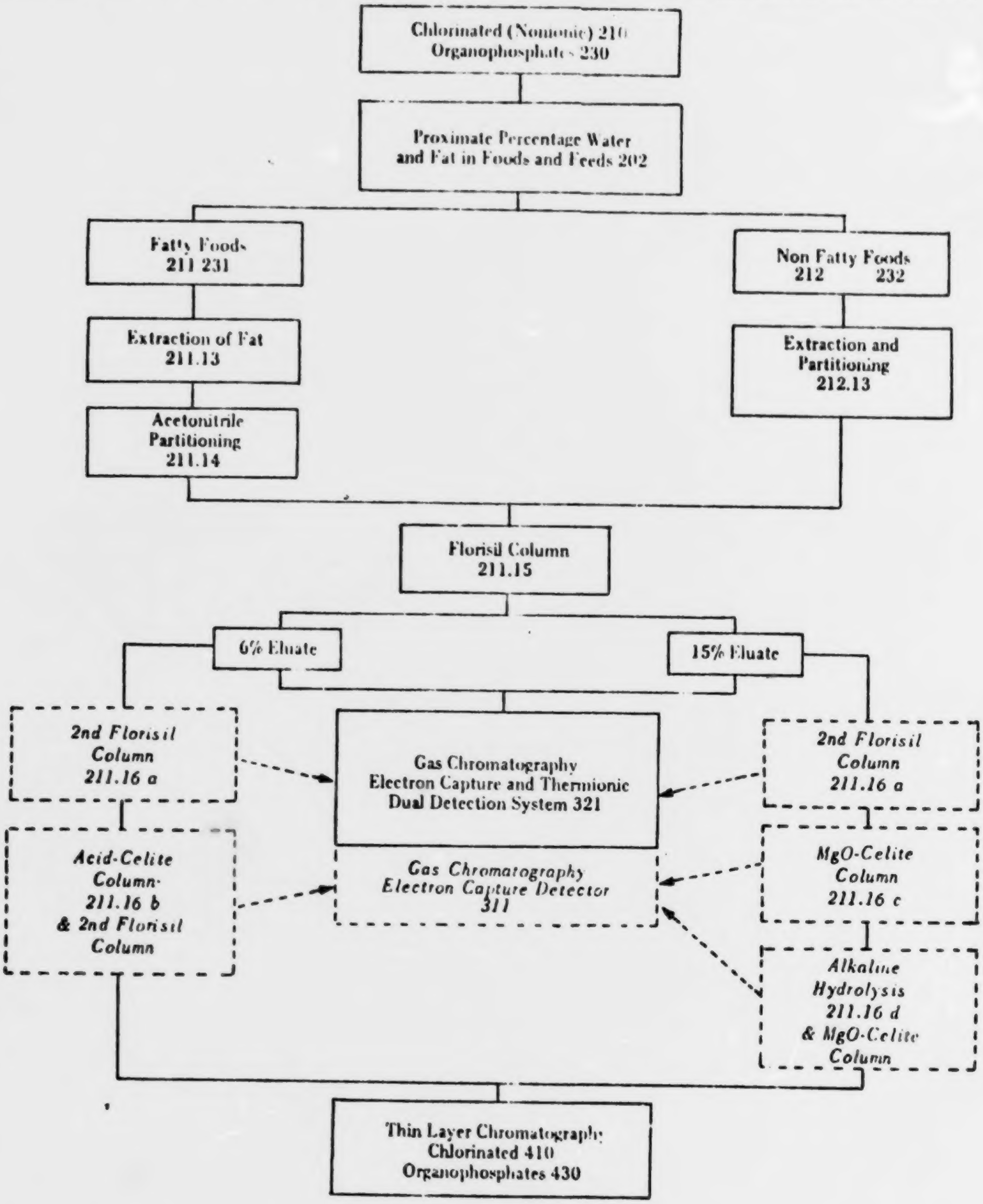
* The numbers refer to the decimal numbering system of PAM. Chapter numbers also refer to PAM.

** Scheme 162 is presented in Figure 2.

Source: PAM, 1971.

Figure 3

ANALYTICAL SCHEME FOR CHLORINATED (NONIONIC) AND ORGANOPHOSPHATE RESIDUES*


Chlorinated (Nonionic) 210
Organophosphates 230
Proximate Percentage Water and Fat in Foods and Feeds 202
Fatty Foods 211 231
Non Fatty Foods 212 232
Extraction of Fat 211.13
Acetonitrile Partitioning 211.14
Extraction and Partitioning 212.13
Florisil Column 211.15
6% Eluate
15% Eluate
2nd Florisil Column 211.16 a
Gas Chromatography Electron Capture and Thermionic Dual Detection System 321
2nd Florisil Column 211.16 a
Acid-Celite Column 211.16 b & 2nd Florisil Column
Gas Chromatography Electron Capture Detector 311
MgO-Celite Column 211.16 c
Alkaline Hydrolysis 211.16 d & MgO-Celite Column
Thin Layer Chromatography Chlorinated 410 Organophosphates 430

* The numbers refer to the decimal numbering system of PAM. The primary analytical scheme is in bold type. Additional cleanup and/or quantitation schemes are in italics.

Source: PAM, 1971.

Potassium Chloride Thermionic Detector

Column packing	Retention time relative to parathion (ratio)	Response (mg for 1/2 FSD*)
10% DC-200 on Chromosorb W-HP (or Gas Chrom Q)	1.00	2
15% QF-1 + 10% DC 200 on Chromosorb W-HP (or Gas Chrom Q)	1.00	2

---

* FSD = Full scale deflection.
** AFS = Amps, full scale.

The PAM does not provide response data for flame photometric detectors. However, this type of detector is now widely used for the analysis of organophosphate residues, primarily because of the high degree of specificity in detecting phosphorus compounds. A review of these and other detectors is provided by Zweig and Sherma (1972)[1].

Residue Analysis Principles

Both AOAC methods manual and PAM (Vol. II) describe methods for the specific analysis of parathion residues. Zweig and Sherma[2] have provided a review of specific residue analytical methods for parathion.

AOAC Method (Official Final Action) - According to the AOAC method[3] for specific analysis of parathion residues, parathion is extracted with benzene or 2-propanol-benzene, and the strip solution is clarified.

1/ Zweig and Sherma, op. cit., p. 205 (1972).
2/ AOAC, op. cit., p. 508 (1970).
3/ Zweig and Sherma, op. cit., p. 445 (1972).

Parathion is brought into aqueous solution and simultaneously reduced to its amine with zinc and hydrochloric acid. The amine is diazotized and coupled with N-(1-naphthyl)-ethylenediamine to form a colored compound which is analyzed spectrophotemetrically.

The practical working range for the Beckman DU spectrophotometer is 0 to 200 ug parathion.

The method has been employed for parathion residues in a wide variety of fruits and vegetables.

PAM Methods - PAM (1967) lists three methods for specific residue analysis. The first two methods have been "tested in varying degrees and are considered reliable without further validation for the product applications indicated." The third method has not been "thoroughly tested through interlaboratory studies."

The First Method - This method refers to the PAM procedure for organophosphate (PAM, 1971); this procedure is summarized in the "Multi-Residue Methods" section of this Appendix.

The Second Method - This method refers to an AOAC procedure (AOAC, 1965)[1]; A summary of an updated AOAC method is described in the preceding section.

According to PAM (Volume II), the AOAC method is generally applicable to all products excluding fats and oils.

The sensitivity is 0.2 ppm.

Cole crops develop an interfering pink color, and blank values as high as 1 ppm and over are not uncommon with some of these crops (Rolston and Walton, 1963[2]).

Aniline and some aniline analogues interfere. Methyl anthranilate, which is found in grapes and citrus fruits, interferes. The amount of methyl anthranilate and hence the extent of interference differs with the maturity and variety of the fruit. (Taschenberg and Avens, 1960[3].

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[1] AOAC, op. cit., (1965).

[2] Rolston, L. H. and R. R. Walton, "Parathion Residues in Greens," J. Econ. Entomol., 56:169-172 (1963).

[3] Taschenberg, E. F. and A. W. Avens, "Parathion and EPN Residue Studies on Concord Grapes," J. Econ. Entomol., 53:441-445 (1960).

Taschenberg and Avens used the procedure of Averell and Norris (1948)[1], except that the benzene solution is repeatedly washed with 10% HCl to remove interference. Alternatively, parathion can be determined by difference: the anthranilate is determined before the parathion is reduced and then both compounds are measured together after reduction. The difference in results represents the parathion.

Decomposed leafy vegetables contain decomposition products which are not removed by the clean-up procedure and which yield false results for parathion.[2]

> The Third Method - The third method is a polarographic procedure which can be used as a rapid scanning technique (Gogan, 1963[3]. The method has been tested on apples and several vegetables. Parathion quantities of less than 0.1 ppm have been determined. Methyl anthranilate does not interfere. Methyl parathion, EPN, and PCNB give peaks sufficiently close to the parathion peak as to interfere with determination. Recent studies by the author indicate that the procedure can be extended to fats and oils, except olive oil, where interferences have been encountered.

Formulation Analysis Principles

Formulation analysis procedures for parathion are described in AOAC "Methods of Analysis" (1970). Additional information has been provided by the Technical Service Division, Office of Pesticide Programs of EPA.

AOAC Methods (Official Final Action) - The two AOAC methods are volumetric (applicable to all forms of technical parathion) and colorimetric (applicable to dusts and wettable powders).

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[1] Averell, P. R. and M. V. Norris, "Estimation of Small Amounts of o, o-diethyl o-(p-nitrophenyl)thiophosphate," Anal. Chem., 20(8): 753-756 (August 1948).

[2] Dow, M. L. (Food and Drug Administration), Personal communication to PAM editors (October 1961).

[3] Gogan, R. J., "Application of Oscillographic Polarography to the Determination of Organophosphorus Pesticides, II. A Rapid Screening Procedure for the Determination of Parathion in Some Fruits and Vegetables," J. Assoc. Off. Agr. Chem., 46(2):216-222 (1963).

The volumetric method[1] This involves a potentiometric titration of the parathion amine (produced by reduction with zinc and hydrochloric acid) with sodium nitrite. p-Nitrophenol in parathion is initially removed by extraction (aqueous sodium carbonate) and determined by ultraviolet spectrophotometry.

The colorimetric method[2] This involves an extraction of parathion with alcohol followed by a hydrolysis of parathion with potassium hydroxide to form potassium p-nitrophenate which is determined by ultraviolet spectrophotometry.

EPA Method - In addition to the AOAC formulation methods, the Technical Service Division of EPA employs a high-pressure liquid chromatographic procedure for parathion analysis. The method is described as follows[3]:

1. Reagents -

   a. Methanol, ACS
   b. Parathion standard of known purity.

2. Operating Conditions for Liquid Chromatograph - UV detector at 254 mm. Operating conditions must be determined for the individual liquid chromatograph to achieve optimum sensitivity and resolution.

3. Procedure -

   a. Standard preparation - Weigh 0.3 g parathion standard into a 100 ml volumetric flask, dissolve, and make to volume with methanol.

   b. Sample preparation - Weigh a portion of sample equivalent to 0.3 g parathion into a glass-stoppered flask, add 100 ml methanol, and shake well.

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[1] AOAC, op. cit., p. 114 (1970).

[2] AOAC, op. cit., p. 115 (1970).

[3] Bontoyan, Warren R., TSD, OPP, EPA, Personal Communication to Dr. Alfred Meiners (September 1974).

4. Determination - Using a high-pressure liquid syringe, alternately inject three 5 ul portions each of standard and sample solutions. Measure the peak area for each peak and calculate the average for both standard and sample. Adjustments in attenuation or amount injected may have to be made to give convenient size peaks.

5. Calculation -

$$\% \text{ parathion} = \frac{(\text{avg area sample})(\text{ul std})(\text{ug/ul std})(\text{purity of std})(100)}{(\text{avg area std})(\text{ul sample})(\text{ug/ul sample})}$$

(This method was developed by Elmer H. Hayes of the Beltsville Chemistry Laboratory, Technical Services Division, Chemical and Biological Investigations.)

Composition and Formulation

The most common formulations of parathion are: (a) wettable powder concentrates (15 to 25%); (b) ready-to-use dry dust mixtures (1 to 10%); (c) emulsifiable concentrates (2 to 8 lb/gal); (d) granules (2 to 25%).

Wettable Powders - Parathion is often formulated as a wettable powder containing 15 to 25% active material. Certain clays perform very well as an absorbent carrier in wettable powder formulations. In addition to the clay carrier, up to 5% of a wetting agent and a dispersing agent are required in the formulation. After making the final blend, the formulation must be ground to a very fine particle size to obtain maximum dispersion in the field spray mixture.

Dusts - Dry dust concentrates may be prepared by impregnating certain absorbent clays with parathion. Typical dry dust concentrates are:

	20% (lb)	25% (lb)
Absorbent clay	79.4	74.3
Parathion (98.5%)	20.6	25.7
	100.0	100.0

Dry 1 to 2.5% field strength dusts may be prepared by diluting concentrates to the desired strength. Secondary type diluents are used in combination to produce the desired bulk density and flow characteristics.

Many formulators prepare field strength dusts by direct impregnation. This is accomplished in the same manner as preparing a concentrate except the mixer is first charged with the desired combination of diluents. Typical direct impregnation formulations are:

	1% (lb)	2% (lb)
Diluents	98.8	97.8
Parathion (98.5%)	1.2	2.2
	100.0	100.0

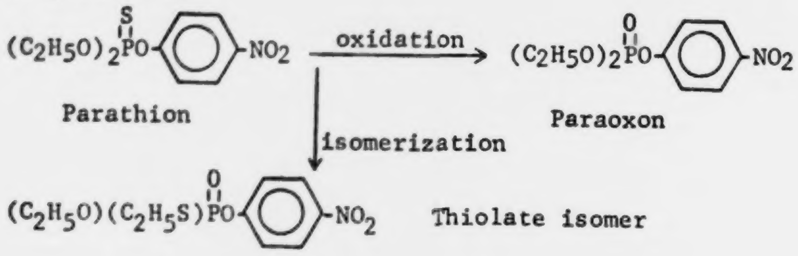
Emulsifiable Concentrates - Parathion can be formulated into many emulsifiable concentrates. Table 2 has four suggested formulations.

Granules - Large quantities of parathion granules are used to protect crop plant roots from soil insects such as rootworms and wireworms. The amount of active ingredient in such formulations varies from 4 to 25% depending on crop, insect and method or time of application.

Granules from calcined or uncalcined clays are the most widely used carriers for parathion. Calcined granules are preferred because of their lower susceptibility to dust formation through attrition.

Chemical Properties, Degradation Reactions and Decomposition Processes

Parathion is a specific chemical compound, O,O-diethyl O-*p*-nitrophenyl phosphorothioate (*Chemical Abstracts* nomenclature). The biological effects of parathion are intimately associated with two other compounds, paraoxon (O,O-diethyl O-*p*-nitrophenyl phosphate) and a thioate isomer of parathion (O,S-diethyl O-*p*-nitrophenyl phosphorothiolate). As indicated below, these compounds are formed by oxidation and isomerization of parathion.


$(C_2H_5O)_2P(S)O$-C_6H_4-NO_2
Parathion
oxidation
$(C_2H_5O)_2P(O)O$-C_6H_4-NO_2
Paraoxon
isomerization
$(C_2H_5O)(C_2H_5S)P(O)O$-C_6H_4-NO_2
Thiolate isomer

Compounds of this kind are toxic primarily because they react with acetylcholinesterase. The toxicity of parathion depends entirely upon its oxidative conversion *in vivo* to paraoxon. Parathion is oxidized enzymatically to paraoxon, but can also be oxidized to paraoxon by the chemical action of atmospheric oxygen. The atmospheric oxidation is

Table 2. SUGGESTED FORMULATIONS FOR PARATHION EMULSIFIABLE CONCENTRATES

Ingredients	% by Wt	Lb/Gal
	2 lb/gal	
Parathion (98.5%)	25.69	2.030[a]
Xylene	69.31	5.479
Emulsifier	5.00	0.395
	100.00	7.904
	4 lb/gal	
Parathion (98.5%)	47.23	4.061[b]
Xylene	45.27	3.892
Emulsifier	7.50	0.645
	100.00	8.598
	6 lb/gal	
Parathion (98.5%)	78.41	7.500[c]
Xylene	14.09	1.348
Emulsifier	7.50	0.717
	100.00	9.565
	8 lb/gal	
Parathion (98.5%)	80.27	8.122[d]
Emulsifier	19.73	1.996
	100.00	10.118

Source: Monsanto Company, Agricultural Division, "Parathion and Methyl Parathion"Technical Bulletin No. AG-1b," St. Louis, Missouri (undated).

a/ Equivalent to 2.0 lb of 100% parathion
Specific Gravity at 25/15.6°C. 0.9485
Solution Point, °C. Cannot Freeze

b/ Equivalent to 4.0 lb of 100% parathion
Specific Gravity at 25/15.6°C. 1.0318
Solution Point, °C. Cannot freeze

c/ Equivalent to 6.0 lb of 100% parathion
Specific Gravity at 25/15.6°C. 1.1478
Solution Point, °C. Cannot freeze

d/ Equivalent to 8.0 lb of 100% parathion
Specific Gravity at 25/15.6°C. 1.2142
Solution Point, °C. Cannot freeze

accelerated by ultraviolet radiation (Cook and Pugh, 1957;[1] Frawley et al., 1958[2]). Paraoxon is much more toxic than parathion. The formation of paraoxon has been postulated as the cause of observed toxicities much higher than expected from the observed concentrations of parathion (Whalley et al., 1964[3]). Paraoxon reacts 10,000 times faster with acetylcholinesterase than does parathion (Heath, 1961[4]).

The thiolate isomer of parathion also reacts with acetylcholinesterase 10,000 times faster than parathion. The isomerization reaction does not proceed in vivo, but does take place in storage. The presence of the contaminating thiolate isomer has invalidated many publications on the biochemical and toxic properties of parathion (Heath, 1961).

The Effect of Sunlight and Ultraviolet Radiation - Parathion was one of the first organic phosphorus compounds whose anticholinesterase activity was shown experimentally to increase during exposure to UV light and sunlight (Payton, 1953[5]). Subsequent work by Cook (1955[6]) and Cook and Pugh (1957) indicated that the toxicity of parathion decreased under UV light, but the in vitro anticholinesterase activity increased as the result of the formation of more polar products. Frawley et al. (1958) found that the exposure of parathion to UV radiation resulted in a mixture of compounds including parathion, paraoxon and oxidation and degradation products. A study by Mitchell (1961[7]) indicated that most organic phosphorus compounds break down to form a wide variety of new compounds on irradiation, but no attempt was made to characterize them. Studies by Koivistoinen and Meriläinen (1962)[1/] demonstrated that both UV radiation and sunlight changed parathion to several cholinesterase inhibitors. On the basis of chromatographic behavior, the metabolites were identified as paraoxon and the S-ethyl (thiolate) and S-phenyl isomers of parathion, together with unknown products (Figure 2). Gar and Kapiani (1956)[2/] and El-Refai and Hopkins (1966)[3/] reported the production of similar mixtures by UV radiation. When methyl parathion was exposed to both UV radiation and sunlight, only the methyl homologue of paraoxon was identified (Koivistoinen and Meriläinen, 1962).

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[1] Cook, J. W., and N. D. Pugh, "Quantitative Study of Cholinesterase-Inhibiting Decomposition Products of Parathion Formed by Ultraviolet Light," J. Assoc. Offic. Agr. Chem., 40:277-281 (1957).

[2] Frawley, J. P., J. W. Cook, J. R. Blake, and O. G. Fitzhugh, "Effect of Light on Chemical and Biological Properties of Parathion," J. Agr. Food Chem., 6(1):28-30 (January 1958).

[3] Whalley, P. J., R. H. Adams, and B. Combes, J. Amer. Med. Assoc., 189(5): 357 (1964).

[4] Heath, D. F., Organophosphorus Poisons, New York: Pergamon Press (1961).

[5] Payton, J., "'Parathion' and Ultraviolet Light," Nature, 171(4347):355-356 (21 February 1953).

[6] Cook, J. W., "Paper Chromatography of Some Organic Phosphate Insecticides. V. Conversion of Organic Phosphates to in vitro Cholinesterase Inhibitors by N-Bromosuccinimide and Ultraviolet Light," J. Assoc. Offic. Agr. Chem., 38:826-832 (1955).

[7] Mitchell, L. C., "Separation and Detection of Eleven Organophosphate Pesticides by Paper Chromatography," J. Assoc. Offic. Agr. Chem., 44:643-712 (1961).

Joiner et al. (1971)[4/] have probably performed the most extensive research to date on photodegradation products of parathion. They subjected parathion to high-intensity ultraviolet radiation under artificial and natural conditions for periods up to 35 days. They detected 12 irradiation products, seven of which had not previously been observed as irradiation products. These products are listed in Table 3.

In a recent study of the photochemical degradation of parathion, O,O,S-triethylthiophosphate was identified as the major product of the photolysis in aqueous tetrahydrofuran or ethanol. Minor products were O,O,O-triethylthiophosphate, paraoxon, and triethylphosphate, which was formed by secondary photolysis of paraoxon (Grunwell and Erickson, 1973[5/]).

Hydrolysis - Hydrolysis is perhaps the most important degradation reaction of organic phosphorus insecticides, because hydrolysis virtually destroys all insecticidal activity.

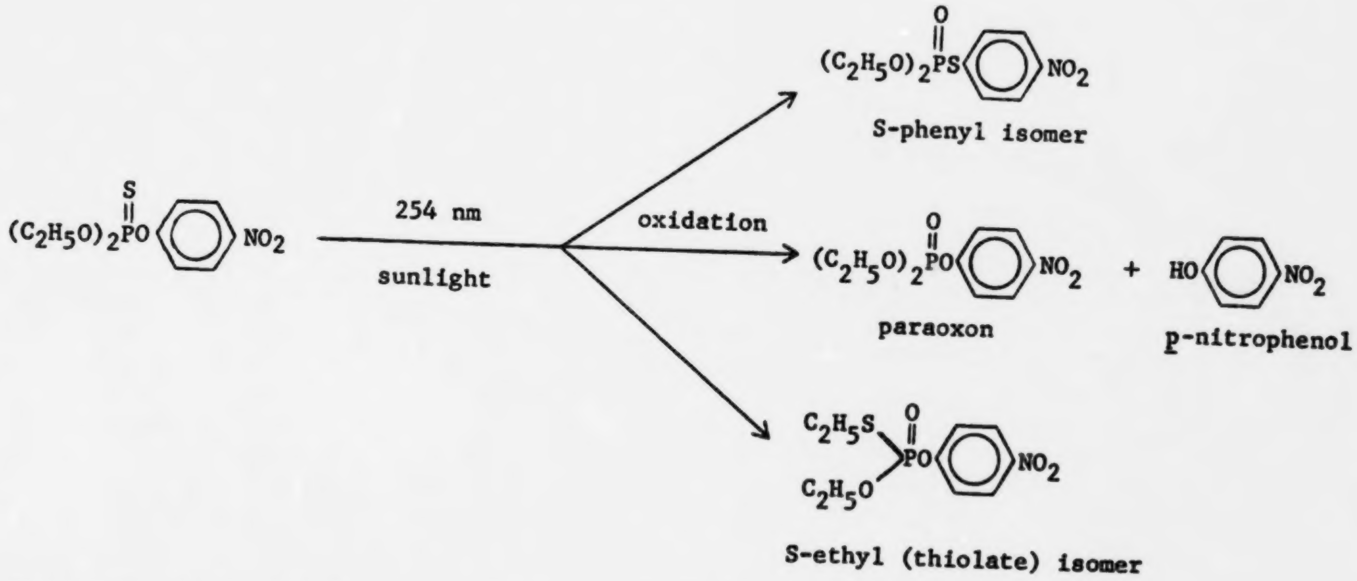
[1/] Koivistoinen, P., and M. Meriläinen, "Paper Chromatographic Studies on the Effect of Ultraviolet Light on Parathion and Its Derivatives," Acta Agr. Scand., 12:267-276 (1962).

[2/] Gar, K. A., and R. Y. Kapiani, Proclamation of the International Conference of Peaceful Uses of Atomic Energy (1955), 12:185 (1956).

[3/] El-Refai, A., and T. L. Hopkins, "Parathion Absorption, Translocation, and Conversion to Paraoxon in Bean Plants," J. Agr. Food Chem., 14(6): 588-592 (November-December 1966).

[4/] Joiner, R. L., H. W. Chambers, and K. P. Baetcke, "Toxicity of Parathion and Several of Its Photoalteration Products to Boll Weevils," Bull. Environ. Contam. Toxicol., 6(3):220-224 (1971).

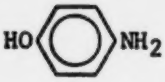
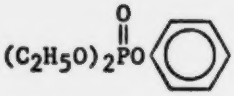
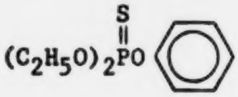
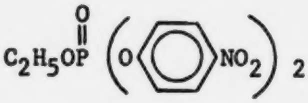
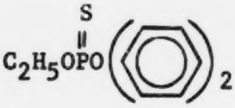
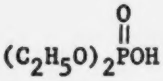
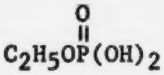
[5/] Grunwell, J. R., and R. H. Erickson, "Photolysis of Parathion (O,O-Diethyl-O-(4-nitrophenylthiophosphate). New Products," J. Agr. Food Chem., 21(5):929-931 (September-October 1973).


254 nm
sunlight
oxidation
S-phenyl isomer
paraoxon
p-nitrophenol
S-ethyl (thiolate) isomer

Adapted from Dauterman, W. C., "Biological and Nonbiological Modifications of Organophosphorus Compounds," Bulletin of the World Health Organization, 44(1-2-3):144 (1971).

Figure 4. Major photodegradation products of parathion.

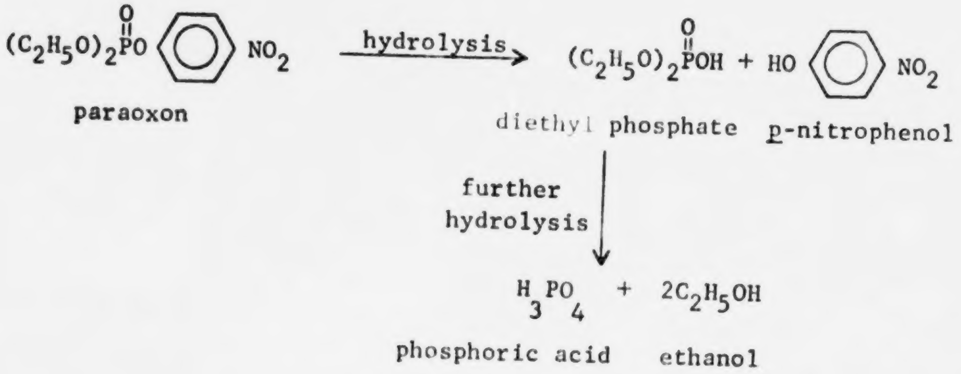
Table 3. OTHER PHOTODEGRADATION PRODUCTS OF PARATHION

p-Aminophenol	
Diethyl phenyl phosphate	
Diethyl phenyl phosphorothioate	
Ethyl bis(p-nitrophenyl) phosphate	
Ethyl bis(p-nitrophenyl) phosphorothioate	
Diethyl phosphate	 $(C_2H_5O)_2\overset{\overset{O}{\|}}{P}OH$
Monoethyl phosphate	 $C_2H_5O\overset{\overset{O}{\|}}{P}(OH)_2$

Data from: Joiner, R. L., Diss. Abstr. Int., 32:4169B (1971/1972).

The extent of hydrolytic stability is, in general, related to the electronic characteristics of the substituents attached to the phosphorus atom. Thus, the replacement of a phosphorus-bound oxygen atom with a sulfur atom usually increases the resistance of the molecule to hydrolysis. This fact probably accounts for the fact that the chemical structures of most commercial organophosphate insecticides contain sulfur.

In parathion (and in methyl parathion) there are three potential groups which may be hydrolyzed from the phosphorus portion of the molecule. However, only one, the p-nitrophenol group, is of practical significance; the hydrolysis of the most labile group greatly increases the hydrolytic stability of the remaining groups. For example, paraoxon has a half-life of 9.2 days at 25°C and pH 10, diethyl phosphate, even at 100°C and pH 10 has a calculated half-life of 5 billion days (Heath, 1956;[1] Vernon, 1957[2]).



Parathion is relatively resistant to hydrolysis. The stability of parathion in water is related to pH and temperature. Stable below pH 7, it hydrolyzes rapidly in alkaline solution. The rate of hydrolysis also increases appreciably with increases in temperature. Fifty percent hydrolysis of parathion is achieved in 2.7 hr at pH 9 and 70°C, but 17 to 20 hr are required at pH 1. At pH 1 to 5, 690 days are required at 20°C (Lawless et al., 1973[3]).

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[1] Heath, D. F., "The Effects of Substituents on the Rates of Hydrolysis of Some Organophosphorus Compounds. I. Rates in Alkaline Solution," J. Chem. Soc., pp. 3796-3804 (1956).

[2] Vernon, C. A., Chem. Soc. (London) Spec. Publ., 8:17 (1957).

[3] Lawless, E. W., T. L. Ferguson, and A. F. Meiners (Midwest Research Institute), Methods for the Disposal of Spilled and Unused Pesticides (Draft), for Environmental Protection Agency, Contract No. 68-01-0098 (1973).

In laboratory studies, the half-life of parathion in ethanol-pH 6.0 buffer solution (20:80) at 70°C was shown to be 43 hr (Ruzicka et al., 1967[1]). With Thames River water (pH 8.0, total hardness as $CaCO_3$ 314 mg) substituted for buffer solution, the half-life was 65 hr; with Irthing River water (pH 7.5, total hardness as $CaCO_3$ 42 mg) substituted, the half-life was 68 hr. Hydrolysis under these conditions was a pseudo-first-order reaction.

In another hydrolysis rate study, 31.2% of parathion remained after 4 weeks in neutral water (Cowart et al., 1971[2]). Studying the persistence of parathion, Mulla (1963)[3] found that, in the laboratory at pH 8, 35°C and a light intensity of 64 ft-candles, parathion remained undegraded after 96 hr; in the field, however, all the insecticide was lost within 72 hr. The more rapid disappearance in the field studies was attributed to wind action, breakdown by microbes, the presence of organic matter, and complex edaphic factors such as absorption by soil colloids.

The Effect of Heat - Metcalf and March (1953)[4] found that when parathion was heated at 150°C for 24 hr, 80 to 90% decomposition resulted and degradation products were formed. Five of the compounds were identified: parathion, paraoxon, *p*-nitrophenol, bis(*p*-nitrophenyl) thionophosphate, and--the principal product--S-ethyl parathion.

Heating most insecticidal organophosphates above 200°C results in decomposition. According to the National Agricultural Chemicals Association (1968),[5] parathion should not be heated above 100°C. When heated to decomposition, parathion emits highly toxic fumes of nitrogen oxides, phosphorus, and sulfur compounds (National Agricultural Chemicals Association, 1968).

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[1] Ruzicka, J. H., J. Thomson, and B. B. Wheals, "The Gas Chromatographic Determination of Organophosphorus Pesticides - Part II. A Comparative Study of Hydrolysis Rates," *J. Chromatog.*, 31(1):37-47 (November 1967).

[2] Cowart, R. P., F. L. Bonner, and E. A. Epps, Jr., "Rate of Hydrolysis of Seven Organophosphate Pesticides," *Bull. Environ. Contam. Toxicol.*, 6(3):231-234 (1971).

[3] Mulla, M. S., "Persistence of Mosquito Larvicides in Water," *Mosquito News*, 23(3):234-237 (September 1963).

[4] Metcalf, R. L., and R. B. March, "The Isomerization of Organic Thiophosphate Insecticides," *J. Econ. Entomol.*, 46:288-294 (April 1953).

[5] National Agricultural Chemicals Association, *Safety Guide for Warehousing Parathions*, Washington, D.C. (1968).

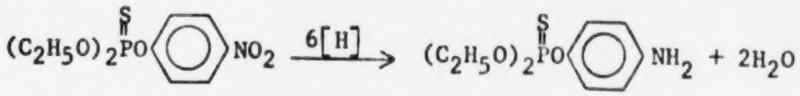
The hazards from fires involving organic phosphorus insecticides have been investigated and it was concluded that: first, most of the insecticide is destroyed by decomposition before it can evaporate; second, over 90% of the evaporating insecticide is destroyed by the flames; and third, the evaporating portion is considerably diluted by the time it reaches anyone (Smith and Ledbetter, 1971[1]).

Oxidation - Several chemical oxidizing agents are capable of replacing the sulfur atom in parathion with an oxygen atom. Koivistoinen and Meriläinen (1962) showed that when parathion was exposed as a thin film, trace amounts of paraoxon were formed even in the absence of light.

A recent publication (Gunther et al., 1970[2]) pointed out that parathion can be rapidly and conveniently oxidized to paraoxon by means of ozone (20 to 40% conversion).

Gomma and Faust (1971)[3] noted that chlorine or potassium permanganate would convert dilute solutions of parathion in water to paraoxon.

Reduction - Reducing agents (for example, metals in acid medium) convert parathion to the corresponding amino compound, aminoparathion (Melnikov, 1971[4]).



The product, 0,0-diethyl 0-4-aminophenyl thiophosphate, is nontoxic to animals and does not have an insecticidal effect.

Occurrence of Parathion Residues in Food and Feed Commodities

The Food and Drug Administration, Department of Health, Education, and Welfare, monitors pesticide residues in the nation's food supply through two programs. One program, commonly known as the "total diet program," involves the examination of food ready to be eaten. This investigation measures the amount of pesticide chemicals found in a high-consumption varied diet. The samples are collected in retail markets and prepared for consumption before analysis. The other program involves the examination of large numbers of samples, obtained when lots are shipped in interstate commerce, to determine compliance with tolerances. These analyses are complemented by observation and investigations in the growing areas to determine the actual practices being followed in the use of pesticide chemicals.

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[1] Smith, W. M., Jr., and J. O. Ledbetter, "Hazards from Fires Involving Organophosphorus Insecticides," Amer. Ind. Hyg. Assoc. J., 32(7):468-474 (July 1971).

[2] Gunther, F. A., D. E. Ott, and M. Ittig, "The Oxidation of Parathion to Paraoxon. II. By Use of Ozone," Bull. Environ. Contam. Toxicol., 5(1):87-94 (1970).

[3] Gomma, H. M., and S. D. Faust, "Chemical Oxidation of Organic Pesticides in Aquatic Environments," Paper No. 48, 161st American Chemical Society Meeting, Los Angeles, California (29 March-12 April 1971).

[4] Melnikov, N. N., Chemistry of Pesticides, Springer-Verlag, New York, pp. 324-329 (1971).

A majority of the samples collected in these programs are categorized as "objective" samples. Objective samples are those collected where there is no suspicion of excessive residues or misuse of the pesticide chemicals. All samples of imported foods and fish are categorized as "objective" samples even though there may be reason to believe excessive residues may be found on successive lots of these food categories.

Market-basket samples for the total diet studies are purchased from retail stores, bimonthly, in five regions of the United States. A shopping guide totaling 117 foods for all regions is used, but not all foods are represented in all regions because of differences in regional dietary patterns. The food items are separated into 12 classes of similar foods and prepared for consumption by dietitians in institutional kitchens. After preparation, the food items are composited into 12 classes of similar foods (e.g., dairy products; meat, fish and poultry; legume vegetables; and garden fruits) for more reliable analysis and to minimize the dilution factor. Each class in each sample is a "composite." The food items and the proportion of each used in the study were developed in cooperation with the Household Economics Research Division, USDA, and represents the high-consumption level of a 16- to 19-year-old male. Each sample represents a 2-week supply of food.

Surveillance samples are generally collected at major harvesting and distribution centers throughout the U.S. and examined in 16 FDA district laboratories. Some samples may be collected in the fields immediately prior to harvest. Surveillance samples are not obtained in retail markets. Samples of imported food are collected when offered for entry into the United States.

The results obtained during the 5-year period, June 1964 to April 1969, are compared in Table 4 with the acceptable daily intake (ADI)

Table 4. DIETARY INTAKE OF PARATHION AND TOTAL ORGANOPHOSPHATES

	Milligrams/kilogram body weight/day total diet studies					
Compounds	1964-1965	1965-1966	1966-1967	1967-1968	1968-1969	5-Year average
Parathion	0.000001	0.000005	0.00001		0.00001	0.00001
Total organophosphates		0.00014	0.00025	0.00007	0.00023	0.0002

Data from Duggan, R. E., G. Q. Lipscomb, E. L. Cox, R. E. Heatwole, and R. C. King, "Pesticide Residue Levels in Foods in the United States from 1 July 1963 to 30 June 1969," Pest. Monit. J., 5(2):73-212 (September 1971).

established by the Food and Agricultural Organization, World Health Organization (FAO/WHO) Expert Committee (FAO/WHO, 1970[1]). The amount of parathion and total organophosphates calculated from this high-consumption diet, approximately twice that consumed by a normal individual, are well below the daily intake regarded as safe by the FAO/WHO Expert Committee (Duggan et al., 1971).[2]

Table 5 (Duggan et al., 1971) compares the incidence and daily intake in milligrams of parathion found in these samples for each of the 5 years.

Table 5. AVERAGE INCIDENT AND DAILY INTAKE OF PARATHION

1965-1966		1966-1967		1967-1968		1968-1969	
Percent positive composites[a]	Daily intake (mg)	Percent positive composities[b]	Daily intake (mg)	Percent positive composites	Daily intake (mg)	Percent positive composites	Daily intake (mg)
1.0	< 0.001	1.4	0.001	0.6	< 0.001	3.3	< 0.001

[a] 312 composites examined.
[b] 360 composites examined.

The results of the FDA analytical studies are tabulated for the following food classes:

- Dairy Products
- Large Fruits
- Small Fruits
- Grains and Cereals (Human)
- Leaf and Stem Vegetables
- Vine and Ear Vegetables
- Root Vegetables
- Beans
- Red Meat
- Poultry
- Eggs
- Fish
- Shellfish
- Grains (Animal)
- Infant and Junior Foods
- Tree Nuts
- Vegetable Oil Products

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[1] FAO/WHO, Food and Agricultural Organization of the United Nations/World Health Organization, *1969 Evaluations of Some Pesticide Residues in Food*, *The Monographs*, Geneva (1970).

[2] Duggan, R. E., G.Q. Lipscomb, E. L. Cox, R. E. Heatwole, and R. C. King, "Pesticide Residue Levels in Foods in the United States from 1 July 1963 to 30 June 1969," *Pest. Monit. J.*, 5(2):73-212 (September 1971).

The most recently available analytical data are presented in Table 6 which lists the incidence and ranges of levels for parathion detected in the various food classes. The omission of any food class from the table indicates that no residues were found.

The available data cover the years 1964 to 1969. Limited data are available for the year 1970 (Corneliussen, 1972[1/]), and a complete update on pesticide residue data is expected in the forthcoming September 1974 issue of the Pesticide Monitoring Journal.

Acceptable Daily Intake

The acceptable daily intake (ADI) is defined as the daily intake which, during an entire life-time, appears to be without appreciable risk on the basis of all known facts at the time of evaluation (Lu, 1973[2/]). It is expressed in milligrams of the chemical per kilogram of body weight (mg/kg).

For parathion the ADI is 0.005 mg/kg. This level was set at the 1965 Joint Meeting of the FAO Committee on Pesticides in Agriculture and the WHO Expert Committee on Pesticide Residues (FAO/WHO, 1968[3/]). A joint meeting is held annually and new evidence is considered which would warrant a change in the ADI of any pesticide. The level for parathion has not been changed through 1971 (FAO/WHO,1972[4/]).

In making the evaluation, all available research on parathion concerning its biochemical effects, toxicology, and teratology is considered.

[1/] Corneliussen, P. E., "Residues in Food and Feed: Pesticide Residues in Total Diet Samples (VI)," Pest. Monit. J., 5(4):313-329 (March 1972).

[2/] Lu, F. C., "Toxicological Evaluation of Food Additives and Pesticide Residues and Their 'Acceptable Daily Intakes' for Man: The Role of WHO, in Conjunction with FAO," Residue Rev., 45:81-93 (1973).

[3/] FAO/WHO, Food and Agricultural Organization of the United Nations/World Health Organization, 1967 Evaluations of Some Pesticide Residues in Food, The Monographs, Geneva (1968).

[4/] FAO/WHO, Food and Agricultural Organization of the United Nations/World Health Organization, "Pesticide Residues in Food," Report of the 1971 Joint FAO/WHO Meeting on Pesticide Residues, World Health Organization Tech. Rept. Series No. 502, Geneva (1972).

Table 6. PERCENT DISTRIBUTION OF PARATHION RESIDUES BY FISCAL YEAR IN DIFFERENT QUANTITATIVE RANGES

	Percent distribution of samples							
	Domestic				Imported			
Range ppm	1964-67	1968	1969	Total	1964-67	1968	1969	Total
Large fruits								
No. samples	2,361	1,512	844	4,717	2,018	155	94	2,267
None found	96.86	94.97	93.36	95.63	99.50	92.26	98.94	98.99
Trace-0.03	1.56	2.71	4.38	2.44	0.29	1.29	1.06	0.40
0.04-0.10	0.80	1.52	0.95	1.06	0.09	2.58	-	0.26
0.11-0.50	0.72	0.60	1.18	0.76	0.09	1.94	-	0.22
0.51-1.00	0.04	0.20	0.12	0.11	-	1.29	-	0.09
1.01-1.50	-	-	-	-	-	0.65	-	0.04
1.51-2.00	-	-	-	-	-	-	-	-
Above 2.00	-	-	-	-	-	-	-	-
Small fruits								
No. samples	825	398	384	1,607	123	57	144	324
None found	95.75	95.48	95.57	95.64	99.18	100.00	99.31	99.38
Trace-0.03	2.18	3.77	2.60	2.68	-	-	0.69	0.31
0.04-0.10	1.33	0.50	1.04	1.06	-	-	-	-
0.11-0.50	0.60	0.25	0.78	0.56	0.81	-	-	0.31
0.51-1.00	0.12	-	-	0.06	-	-	-	-
1.01-1.50	-	-	-	-	-	0.65	-	0.04
1.51-2.00	-	-	-	-	-	-	-	-
Above 2.00	-	-	-	-	-	-	-	-
Grains and cereal for human use								
No. samples	2,107	877	581	3,565	20	8	4	32
None found	99.57	98.75	99.14	99.30	100.00	100.00	100.00	100.00
Trace-0.03	0.33	1.14	0.86	0.62	-	-	-	-
0.04-0.10	0.09	-	-	0.06	-	-	-	-
0.11-0.50	-	0.11	-	0.03	-	-	-	-
0.51-1.00	-	-	-	-	-	-	-	-
1.01-1.50	-	-	-	-	-	-	-	-
1.51-2.00	-	-	-	-	-	-	-	-
Above 2.00	-	-	-	-	-	-	-	-
Leaf and stem vegetables								
No. samples	5,780	2,251	1,782	9,813	35	8	19	62
None found	88.14	80.36	75.08	83.99	100.00	100.00	94.74	98.39
Trace-0.03	4.15	9.02	9.60	6.26	-	-	5.26	1.61
0.04-0.10	2.49	5.29	6.34	3.83	-	-	-	-
0.11-0.50	3.94	4.09	7.24	4.58	-	-	-	-
0.51-1.00	0.76	0.89	0.84	0.81	-	-	-	-
1.01-1.50	0.27	0.18	0.39	0.28	-	-	-	-
1.51-2.00	0.06	0.09	0.22	0.10	-	-	-	-
Above 2.00	0.15	0.09	0.28	0.16	-	-	-	-

Table 6. (Concluded)

	Percent distribution of samples							
	Domestic				Imported			
Range ppm	1964-67	1968	1969	Total	1964-67	1968	1969	Total
				Vine and ear vegetables				
No. samples	4,401	1,059	848	5,308	1,040	254	280	1,574
None found	98.44	94.62	94.69	97.08	95.09	83.46	75.00	80.64
Trace-0.03	0.88	3.97	3.18	1.87	2.88	11.81	18.93	7.18
0.04-0.10	0.38	1.04	1.42	0.68	1.44	3.94	2.86	2.10
0.11-0.50	0.23	0.38	0.71	0.34	0.48	0.79	2.86	0.95
0.51-1.00	0.02	-	-	0.02	0.09	-	0.36	0.13
1.01-1.50	-	-	-	-	-	-	-	-
1.51-2.00	0.02	-	-	0.02	-	-	-	-
Above 2.00	-	-	-	-	-	-	-	-
				Root vegetables				
No. samples	5,425	1,758	1,060	8,243	165	66	89	320
None found	99.26	97.50	97.55	98.67	100.00	100.00	95.51	98.75
Trace-0.03	0.55	1.59	1.70	0.92	-	-	2.25	0.62
0.04-0.10	0.14	0.34	0.66	0.25	-	-	2.25	0.62
0.11-0.50	0.01	0.40	0.09	0.11	-	-	-	-
0.51-1.00	0.01	0.11	-	0.04	-	-	-	-
1.01-1.50	-	-	-	-	-	-	-	-
1.51-2.00	-	-	-	-	-	-	-	-
Above 2.00	-	0.06	-	0.01	-	-	-	-
				Beans				
No. samples	681	106	81	868	79	27	24	130
None found	98.82	99.06	97.53	98.73	87.34	74.07	66.67	80.77
Trace-0.03	0.29	-	2.47	0.46	6.32	11.11	16.67	9.23
0.04-0.10	0.73	0.94	-	0.69	3.79	11.11	8.33	6.15
0.11-0.50	0.14	-	-	0.12	2.53	3.70	4.17	3.08
0.51-1.00	-	-	-	-	-	-	4.17	0.77
1.01-1.50	-	-	-	-	-	-	-	-
1.51-2.00	-	-	-	-	-	-	-	-
Above 2.00	-	-	-	-	-	-	-	-

Data from Duggan et al., *op. cit.* (1971).

Tolerances

U.S. Tolerances - Section 408 of the Food, Drug and Cosmetic Act, as amended gives procedures for establishing tolerances for pesticide chemicals on raw agricultural commodities. Section 409 applies to food additives, including pesticide chemicals on processed foods. Tolerances are published in the Code of Federal Regulations, Title 40, and in the Federal Register. When both methyl parathion and parathion are present, the tolerances apply to their combined total. A summary of current U.S. tolerances for parathion on raw agricultural commodities is presented in Table 7.

According to Lu (1973), U.S. tolerances which are established should not result in the maximum ADI being reached each day. He gives the following reasons:

1. The tolerance reflects the maximum level of residue resulting from good agricultural practice, but this level is often not reached.
2. The tolerance is based on the assumption that the particular pesticide is used on all food in the class in question, and this is rarely the case.
3. Much of the residue will be lost in storage, processing and cooking.

The tolerances are also based upon the entire product as purchased in the market. However, the product, as purchased, may not be entirely consumed.

Table 7. SUMMARY OF U.S. TOLERANCES FOR PARATHION AND/OR METHYL PARATHION ON RAW AGRICULTURAL COMMODITIES[a/]

ppm	Crop	ppm	Crop	ppm	Crop
0.1 (N)	Almonds	1	Endive (escarole)	0.9 (1) (N)	Potatoes
1.25	Alfalfa fresh	1	Figs	1	Pumpkins
5	Alfalfa hay	0.1 (N)	Filberts	1	Quinces
1	Apples	1	Garlic	1	Radishes (with or without tops)
1	Apricots	1	Gooseberries	1	Radish tops
1	Artichokes	1	Grapes	0.2	Rapeseed
1	Avocados	1	Grass for forage	1	Raspberries
1	Barley	1	Guavas	1	Rice
1	Beans	1	Hops	1	Rutabagas (with or without tops)
1	Beets (with or without tops)	1	Kale	1	Rutabaga tops
1	Beet greens	1	Kohlrabi	0.5 (1) (N)	Rye
1	Blackberries	1	Lettuce	0.1 (1) (N)	Safflower seed
1	Blueberries (huckleberries)	1	Loganberries	0.1 (1) (N)	Sorghum
1	Boysenberries	1	Mangoes	1	Soybean hay
1	Broccoli	1	Melons	0.1	Soybeans
1	Brussels sprouts	1	Mustard greens	1	Spinach
1	Cabbage	0.2	Mustard seed	1	Squash
1	Carrots	1	Nectarines	1	Strawberries
1	Cauliflower	1	Oats	0.1 (1) (N)	Sugar beets
1	Celery	1	Okra	0.1 (1) (N)	Sugarcane
1	Cherries	1	Olives	1	Summer squash
1	Citrus fruits	1	Onions	0.2	Sunflower seed
1	Clover	1	Parsnips (with or without tops)	0.1 (1) (N)	Sweet potatoes
1	Collards	1	Parsnip greens	1	Swiss chard
1	Corn	1	Peaches	1	Tomatoes
1	Corn forage	1	Pea forage	1	Turnips (with or without tops)
0.75	Cottonseed	1	Peanuts	1	Turnip greens
1	Cranberries	1	Pears	1	Vetch
1	Cucumbers	1	Peas	0.1 (1) (N)	Walnuts
1	Currants	0.1 (1) (N)	Pecans	1	Wheat
1	Dates	1	Peppers	1	Youngberries
1	Dewberries	1	Pineapples		
1	Eggplants	1	Plums (fresh prunes)		

Administrative guidelines - none.

Tolerances pending - all interim tolerances above.

(1) - Interim tolerances.

(N) - Negligible residue tolerances.

Source: Reprinted from Pesticide Chemical News Guide of Food Chemical News, Inc., by permission of the publisher, first published 1 May 1974.

[a/] Tolerances apply to combined methyl parathion and parathion residue if both are present.

International Tolerances - Tolerances established by individual nations may be based on recommendations of the FAO/WHO Expert Committee on Food Additives. The Committee evaluates all residue data submitted by interested parties and uses the following criteria (FAO/WHO, 1962,[1]) for making tolerance recommendations:

1. Decide upon the effective level of the food additive under consideration that would be needed in good technological practice.

2. Examine the possible uses and list all the foods in which the food additive might be used.

3. Calculate the daily intake level that might occur if the food additive was used in all the foods for which it might be a useful additive, working on the basis of the average intake of the food materials containing the additive. This average intake for appropriate population groups is obtained from national food consumption surveys.

4. Obtain the necessary information from which to calculate the average body weight of the population group concerned (usually between 50 to 70 kg).

5. From this information, calculate the intake of the additive in milligrams per kilograms of body weight per day.

6. Check the figure against the acceptable intakes given for the substances in the table. If it falls within the unconditional intake zone, the situation is satisfactory and the level proposed may be accepted. If it falls within the conditional intake zone, further scientific advice is required before the level of use proposed is accepted.

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[1] FAO/WHO, Food and Agricultural Organization of the United Nations/ World Health Organization, "Evaluation of the Toxicity of a Number of Antimicrobials and Antioxidants," Sixth Report, Joint FAO/WHO Expert Committee on Food Additives, World Health Organization Tech. Rept. Series No. 228, Geneva (1962).

The validity of criteria for determining tolerances was reaffirmed at the 1966 FAO/WHO, 1967[1]).

The recommendations for parathion tolerances established by the 1969 Joint Meeting of the FAO and WHO (FAO/WHO, 1970) are:

Vegetables (except carrots)	0.7 ppm
Peaches, apricots, other fresh fruits	1.0 ppm
Other fresh fruits	0.5 ppm

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[1] FAO/WHO, Food and Agricultural Organization of the United Nations/ World Health Organization, "Specifications for the Identity and Purity of Food Additives and Their Toxicological Evaluation: Some Emulsifiers and Stabilizers and Certain Other Substances,: 10th Report, Joint FAO/WHO Expert Committee on Food Additives, World Health Organization Tech. Rept. Series No. 373, Geneva (1967).

References

Association of Official Analytical Chemists, Official Methods of Analysis of the Association of Official Analytical Chemists, 11th ed., Washington, D.C. (1970).

Averell, P. R. and M. V. Norris, "Estimation of Small Amounts of O,O-diethyl O-(*p*-nitrophenyl)thiophosphate," Anal. Chem., 20(8):753-756 (August 1948).

Bright, N. F. H., J. C. Cuthill, and N. H. Woodbury, "The Vapor Pressure of Parathion and Related Compounds," J. Sci. Food and Agr., 1:344-348 (November 1950).

Cook, J. W., "Paper Chromatography of Some Organic Phosphate Insecticides. V. Conversion of Organic Phosphates to *in vitro* Cholinesterase Inhibitors by N-Bromosuccinimide and Ultraviolet Light," J. Assoc. Offic. Agr. Chem., 38-826-832 (1955).

Cook, J. W., and N. D. Pugh, "Quantitative Study of Cholinesterase-Inhibiting Decomposition Products of Parathion Formed by Ultraviolet Light," J. Assoc. Offic. Agr. Chem., 40:277-281 (1957).

Corneliussen, P. E., "Residues in Food and Feed: Pesticide Residues in Total Diet Samples (VI)," Pest. Monit. J., 5(4):313-329 (March 1972).

Cowart, R. P., F. L. Bonner, and E. A. Epps, Jr., "Rate of Hydrolysis of Seven Organophosphate Pesticides," Bull. Environ. Contam. Toxicol., 6(3):231-234 (1971).

Dauterman, W. C., "Biological and Nonbiological Modifications of Organophosphorus Compounds," Bulletin of the World Health Organization, 44(1-2-3):144 (1971).

Duggan, R. E., G. Q. Lipscomb, E. L. Cox, R. E. Heatwole, and R. C. King, "Pesticide Residue Levels in Foods in the United States from 1 July 1963 to 30 June 1969," Pest. Monit. J., 5(2):73-212 (September 1971).

Dvornikoff, M. N., et al. (to Monsanto Chemical Company), U.S. Patent No. 2,663,721 (22 December 1953).

El-Refai, A., and T. L. Hopkins, "Parathion Absorption, Translocation, and Conversion to Paraoxon in Bean Plants," J. Agr. Food Chem., 14(6):588-592 (November-December 1966).

FAO/WHO, Food and Agricultural Organization of the United States/World Health Organization, "Evaluation of the Toxicity of a Number of Antimicrobials and Antioxidants," Sixth Report, Joint FAO/WHO Expert Committee on Food Additives, World Health Organization Tech. Rept. Series No. 228, Geneva (1962).

FAO/WHO, Food and Agricultural Organization of the United Nations/World Health Organization, "Specifications for the Identity and Purity of Food Additives and Their Toxicological Evaluation: Some Emulsifiers and Stabilizers and Certain Other Substances," 10th Report, Joint FAO/WHO Expert Committee on Food Additives, World Health Organization Tech. Rept. Series No. 373, Geneva (1967).

FAO/WHO, Food and Agricultural Organization of the United Nations/World Health Organization, 1967 Evaluation of Some Pesticide Residues in Food, The Monographs, Geneva (1968).

FAO/WHO, Food and Agricultural Organization of the United Nations/World Health Organization, 1969 Evaluations of Some Pesticide Residues in Food, The Monographs, Geneva (1970).

FAO/WHO, Food and Agricultural Organization of the United Nations/World Health Organization, "Pesticide Residues in Food," Report of the 1971 Joint FAO/WHO Meeting on Pesticide Residues, World Health Organization Tech. Rept. Series No. 502, Geneva (1972).

Frawley, J. P., J. W. Cook, J. R. Blake, and O. G. Fitzhugh, "Effect of Light on Chemical and Biological Properties of Parathion," J. Agr. Food Chem., 6(1):28-30 (January 1958).

Gar, K. A., and R. Y. Kapiani, Proclamation of the International Conference of Peaceful Uses of Atomic Energy (1955), 12:185 (1956).

Gogan, R. J., "Application of Oscillographic Polarography to the Determination of Organophosphorus Pesticides, II. A Rapid Screening Procedure for the Determination of Parathion in Some Fruits and Vegetables," J. Assoc. Off. Agr. Chem., 46(2):216-222 (1963).

Gomma, H. M., and S. D. Faust, "Chemical Oxidation of Organic Pesticides in Aquatic Environments," Paper No. 48, 161st American Chemical Society Meeting, Los Angeles, California (29 March - 12 April 1971).

Grunwell, J. R., and R. H. Erickson, "Photolysis of Parathion (0,0-Diethyl-0-(4-nitrophenyl)thiophosphate). New Producers," J. Agr. Food Chem., 21(5):929-931 (September-October 1973).

Gunther, F. A., D. E. Ott, and M. Ittig, "The Oxidation of Parathion to Paraoxon, II. By Use of Ozone," Bull. Environ. Contam. Toxicol., 5(1):87-94 (1970).

Heath, D. F., "The Effects of Substituents on the Rates of Hydrolysis of Some Organophosphorus Compounds. I. Rates in Alkaline Solution," J. Chem. Soc., pp. 3796-3804 (1956).

Heath, D. F., Organophosphorus Poisons, New York: Pergamon Press (1961).

Joiner, R. L., H. W. Chambers, and K. P. Baetcke, "Toxicity of Parathion and Several of Its Photoalteration Products to Boll Weevils," Bull. Environ. Contam. Toxicol., 6(3):220-224 (1971).

Joiner, R. L., Diss. Abstr. Int., 32:4169B (1971/1972).

Koivistoinen, P., and M. Meriläinen, "Paper Chromatographic Studies on the Effect of Ultraviolet Light on Parathion and Its Derivatives," Acta Agr. Scand., 12:267-276 (1962).

Lawless, E. W., and T. L. Ferguson (Midwest Research Institute), and R. von Rümker (RvR Consultants), The Pollution Potential in Pesticide Manufacturing, for Environmental Protection Agency, Contract No. 68-01-0142 (January 1972).

Lawless, E. W., T. L. Ferguson, and A. F. Meiners (Midwest Research Institute), Methods for the Disposal of Spilled and Unused Pesticides (Draft), for Environmental Protection Agency, Contract No. 68-01-0098 (1973).

Lu, F. C., "Toxicological Evaluation of Food Additives and Pesticide Residues and Their 'Acceptable Daily Intakes' for Man: The Role of WHO, in Conjunction with FAO," Residue Rev., 45:81-93 (1973).

Melnikov, N. N., Chemistry of Pesticides, Springer-Verlag, New York, pp. 324-329 (1971).

Metcalf, R. L., and R. B. March, "The Isomerization of Organic Thiophosphate Insecticides," J. Econ. Entomol., 46:288-294 (April 1953).

Mitchell, L. C., "Separation and Detection of Eleven Organophosphate Pesticides by Paper Chromatography," J. Assoc. Offic. Agr. Chem., 44: 643-712 (1961).

Monsanto Company, Agricultural Division, "Parathion and Methyl Parathion," Technical Bulletin No. AG-1b, St. Louis, Missouri (undated).

Mulla, M. S., "Persistence of Mosquito Larvicides in Water," Mosquito News, 23(3):234-237 (September 1963).

National Agricultural Chemicals Association, Safety Guide for Warehousing Parathions, Washington, D.C. (1968).

Payton, J., "'Parathion' and Ultraviolet Light," Nature, 171(4347):355-356 (21 February 1953).

The Pesticide Chemical News Guide (1 May 1974).

Rolston, L. H., and R. R. Walton, "Parathion Residues in Greens," J. Econ. Entomol., 56:169-172 (1963).

Ruzicka, J. H., J. Thomson, and B. B. Wheals, "The Gas Chromatographic Determination of Organophosphorus Pesticides - Part II. A Comparative Study of Hydrolysis Rates," J. Chromatog., 31(1):37-47 (November 1967).

Schrader, G. (to Farbenfabriken Bayer), U.S. Patent No. 2,624,745 (6 January 1953).

Smith, W. M., Jr., and J. O. Ledbetter, "Hazards from Fires Involving Organophosphorus Insecticides," Amer. Ind. Hyg. Assoc. J., 32(7):468-474 (July 1971).

Taschenberg, E. G. and A. W. Avens, "Parathion and EPN Residue Studies on Concord Grapes," J. Econ. Entomol., 53:441-445 (1960).

Toy, A. D. F., et al. (to Victor Works), U.S. Patent No. 2,471,464 (31 May 1949).

U.S. Department of Agriculture, Food and Drug Administration, Pesticide Analytical Manual, 2 vols. (1971).

Vernon, C. A., Chem. Soc. (London) Spec. Publ., 8:17 (1957).

Whalley, P. J., R. H. Adams, and B. Combes, J. Amer. Med. Assoc., 189(5):357 (1964).

Zweig, G., and J. Sherma, Analytical Methods for Pesticides and Plant Growth Regulators, Vol. VI: Gas Chromatographic Analysis, Academic Press, New York, New York (1972).

SUBPART II. B. PHARMACOLOGY AND TOXICOLOGY

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This section is concerned with information on the acute, subacute and chronic toxicities of parathion in laboratory and domestic animals. The metabolism of parathion is reviewed; the absorption, distribution, excretion, biotransformation, activation and detoxification of this compound are discussed. The effects of parathion on reproduction, malformation of the young, and behavioral effects are also considered. Reviews of parathion's toxic effects on tissue cultures, and the promotion or tendencies to bring about mutagenesis and oncogenic effects are included, although information on these latter effects is quite limited. The hazards posed by exposure of humans to parathion have been reviewed in relation to acute and subacute toxicity, the symptoms associated with parathion poisoning, the physiological and pharmacological action, the routes of exposure (mainly dermal and inhalation), and the hazards associated with the use of parathion in field operations and in manufacturing plants.

Acute, Subacute and Chronic Toxicity

The information in this subsection is related to the toxicological studies in laboratory and domestic animals.

Toxicity to Laboratory Animals -

Acute oral toxicity - rats - Tables 8 and 9 present the findings of several studies on LD_{50} values for parathion administered in rats by oral, dermal, intraperitoneal, intravenous, intramuscular means and by inhalation. The individual LD_{50} values obtained varied from one study to another. With only one exception, females were found to be more sensitive to oral administration of the insecticide than males. The individual values for reported LD_{50}'s in Table 8 were used to calculate an average LD_{50} value. This average oral LD_{50} was 7.6 mg/kg (range 2.04 to 30.0 mg/kg) for male rats and 3.5 mg/kg (range 1.75 to 6.0 mg/kg) for females.

If data in Table 8 are compared to data reported by Gaines (1960)[1], there is good agreement among researchers on confidence levels for female rats and relatively good agreement on levels for male rats. With females the confidence limits are 3.2 to 4.0 mg/kg, and the average LD_{50} is 3.5 mg/kg. The confidence limits for male rats reported by Gaines are from 10 mg/kg to 17 mg/kg; the computed average from Table 8 is 7.6 mg/kg. One value (30 mg/kg) in Table 8 was not used in computing the average because it was thought to lie too far outside the range of the other values reported. If the 30 mg/kg LD_{50} were included in the computation, the average would then be slightly higher than 9 mg/kg, and this value would be very close to the lower confidence limit reported for male rats by Gaines.

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[1] Gaines, T. G., "The Acute Toxicity of Pesticides to Rats," Toxicol. Appl. Pharmacol., 2:88-99 (1960).

Table 8. ACUTE ORAL TOXICITY OF PARATHION FOR RATS

Measurement (mg/kg unless noted otherwise)	Male	Female	Mixed	Weanlings (male)	Reference
Oral LD_{50}	5.0	1.75	-	-	a/
Oral LD_{50}	13.0	3.60	-	-	b/, c/, d/, e/
Oral LD_{50}	-	4.03	-	-	f/
Oral LD_{50}	-	3.20	-	-	g/
Oral LD_{50}	3.6	-	-	-	h/
Oral LD_{50}	-	-	-	1.5	h/
Oral LD_{50}	-	-	3-6	-	i/
Oral LD_{50}	2.04	-	-	-	j/
Oral LD_{50}	-	-	2.5	-	k/
Oral LD_{50}	8.6	-	-	-	l/
Oral LD_{50}	7.0	4.0	-	1.5	m/
Oral LD_{50}	3.6	-	-	-	n/
Oral LD_{50}	7.5	-	-	-	o/
Oral LD_{50}	5.9	2.70	-	1.75	p/
Oral LD_{50}	12.50	3.50	-	-	a/
Oral LD_{50}	30.00*	3.00	-	-	q/
Oral LD_{50}	15.0	6.00	-	-	r/
Oral LD_{50} $\bar{x}$	7.6	3.5	-	-	
Range	2.04-30.0	1.75-6.0	-	-	
19/20 confidence limits (mg/kg)	10-17	3.2-4.0	-	-	b/
Lowest dose to kill an adult rat					
Oral	10	3.00	-	-	c/
Dermal	10	5.00	-	-	c/

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* Not included in average calculation.

a/ Hazleton, L. W., and E. G. Holland, "Pharmacology and Toxicology of Parathion," Adv. Chem. Series No. 1, 31:31-38 (1950).

b/ Gaines, T. B., *op. cit.* (1960).

c/ Gaines, T. B., "Acute Toxicity of Pesticides," Toxicol. Appl. Pharmacol., 14:515-534 (1969).

d/ Anon., "Toxic Hazards of Pesticides to Man. Report of a Study Group," WHO Tech. Rept. Ser., 114:3-51 (1956).

e/ Technical Development Laboratories, Communicable Disease Center, Clinical Memoranda on Economic Poisons, U.S. Department of Health, Education, and Welfare, Public Health Service, pp. 7-12 and 17-21 (1 April 1956).

f/ Deichmann, W. B., W. Pugliese, and J. Cassidy, "Effects of Dimethyl and Diethyl Paranitrophenyl Thiophosphate on Experimental Animals," AMA Arch. Ind. Hyg. Occup. Med., 5:44-51 (1952).

g/ Edson, E. F., and D. N. Noakes, "The Comparative Toxicity of Six Organophosphorus Insecticides in the Rat," Toxicol. Appl. Pharmacol., 2:523-539 (1960).

h/ Brodeur, J., and K. P. DuBois, "Comparison of Acute Toxicity of Anticholinesterase Insecticides to Weanling and Adult Male Rats," Proc. Soc. Exp. Biol. Med., 114(2):509-511 (November 1963).

i/ Edson, E. F., D. M. Sanderson, and D. N. Noakes, "Acute Toxicity Data for Pesticides (1964)," World Rev. Pest Con., 4:36-41 (1965).

j/ Webb, R. E., C. C. Bloomer, and C. L. Miranda, "Effect of Casein Diets on the Toxicity of Malathion and Parathion and Their Oxygen Analogues," Bull. Environ. Contam. Toxicol., 9(2):102-107 (1973).

k/ DuBois, K. P., and F. K. Kinoshita, "Influence of Induction of Hepatic Microsomal Enzymes by Phenobarbital on Toxicity of Organic Phosphate Insecticides," Proc. Soc. Exp. Biol. Med., 129:699-702 (1968).

l/ Villeneuve, D. C., and W. E. J. Phillips, "The Effect of Acute Ethanol Administration on Parathion Toxicity and *in vitro* Parathion Degradation on the Rat," Can. J. Physiol. Pharmacol., 49:481-483 (1971).

m/ DuBois, K. P., "The Toxicity of Organophosphorus Compounds to Mammals," Bulletin of the World Health Organization, 44:233-240 (1971).

n/ Dalloz, J. P., Delatour, and G. Lorgue, "The Organophosphorus Pesticides," Rev. Med. Vet., Toulouse, 123(10):1356 (1972).

o/ Jacobsen, P. L., R. C. Spear, E. Wei, "Parathion and Diisopropylfluorophosphate (DFP) Toxicity in Partially Hepatectomized Rats," Toxicol. Appl. Pharmacol., 26:314-317 (1973).

p/ Alary, J-G., and J. Brodeur, "Correlation Between the Activity of Liver Enzymes and the LD_{50} of Parathion in the Rat," Can. J. Physiol. Pharmacol., 48:829-831 (1970).

q/ Frawley, J. P., E. C. Hagan, and O. G. Fitzhugh, "A Comparative Pharmacological and Toxicological Study of Organic Phosphate-Anticholinesterase Compounds," J. Pharmacol. Exp. Ther., 105:156-165 (1952).

r/ DuBois, K. P., J. Doull, P. R. Salerno, and J. M. Coon, "Studies on the Toxicity and Mechanism of Action of *p*-Nitrophenyl Diethyl Thionophosphate (Parathion)," J. Pharmacol. Exp. Ther., 95(1):79-91 (1949).

Acute toxicity - rats, routes other than oral - The toxicity of parathion for rats by exposures other than oral (Table 9) has not been studied as thoroughly as by oral toxicity.

The intraperitoneal LD_{50} values range from 3.6 to 7.0 mg/kg for males, and 4 mg/kg was the only value found for females. The dermal LD_{50} for rats was about 21 mg/kg for males with much more variation for females, 6.8 to 100 mg/kg. The LD_{50} value for inhalation toxicity has been reported as 0.0315 mg/liter (4 hr) and 0.115 mg/liter (1 hr). Gaines states that ". . .results and use experience indicates that there is a much closer relationship between dermal LD_{50} values and the occurrence of occupational poisoning than between oral LD_{50} values and occupational poisoning."

It is interesting that the values reported for inhalation toxicity indicate that very low levels of parathion are toxic to rats (LD_{50}-- 0.115 mg/liter - 1 hr; and 0.315 mg/liter - 4 hr).

Subacute and chronic oral toxicity - rats - Only a few studies have been reported in the open literature on subacute and chronic toxicity of parathion in rats. Barnes and Denz (1951)[1/] and Edson and Noakes (1960) conducted relatively long-term studies on toxicity. Under the Edson and Noakes study, rats were fed diets containing parathion for 15 to 16 weeks, at which time survivors were sacrificed, and macroscopic examinations were conducted. Relative organ weights and measurement of cholinesterase activity in red cells, plasma, and whole brain were made. In this study death due to parathion toxicity was reported only in the animals fed at a dietary concentration of 125 ppm, calculated to be equal to an average parathion intake of 15.4 mg/kg/day. Thirty percent mortality (three of 10) occurred in this group. No mortality occurred in those groups of rats fed at dietary levels of 25 ppm (X = 2.4 mg/kg/day) or at 5 ppm (X = 0.52 mg/kg/day).

[1/] Barnes, J. M., and F. A. Denz, "The Chronic Toxicity of p-Nitrophenyl Diethyl Thiophosphate (E. 605): A Long-Term Feeding Experiment with Rats," J. Hyg., 49:430-441 (1951).

Table 9. ACUTE TOXICITY OF PARATHION FOR RATS VIA ROUTES OTHER THAN ORAL

Measurement (mg/kg unless noted otherwise)	Adults			Weanlings (male)	Reference
	Male	Female	Mixed		
IP LD_{50}	7.0	4.0	-	-	a/
IP LD_{50}	-	-	4.5	-	b/
IP LD_{50}	3.6	-	-	1.5	c/
D LD_{50}	21.0	10.9	-	-	d/, e/
D LD_{50}	-	100.0	-	-	f/
D LD_{50}	-	-	50-200	-	g/
D LD_{50}	21.0	6.8	-	-	h/
LC_{50} - 1 hr (mg/ℓ)	0.115	-	-	-	c/
LC_{50} - 4 hr (mg/ℓ)	0.0315	-	-	-	c/

IP LD_{50} - Intraperitoneal injection.

D LD_{50} - Dermal exposure.

LC_{50} - Lethal concentration by inhalation.

a/ DuBois et al., *op. cit.* (1949).

b/ Westermann, E., "Accumulation of Environmental Agents or Their Effects in the Body," *Environ. Res.*, 2:340-351 (1969).

c/ Kimmerle, G., and D. Lorke, "Toxicology of Insecticidal Organophosphates," *Pflanz. Nachr. Bayer*, 21:111-142 (1968).

d/ Anon., *op. cit.* (1956).

e/ Technical Development Laboratories, *op. cit.* (1956).

f/ Edson et al., *op. cit.* (1960).

g/ Edson et al., *op. cit.* (1965).

h/ Gaines, T. B., *op. cit.* (1969).

Pertinent findings from the study by Barnes and Denz (1951) are incorporated into Table 10. No food consumption data was obtained in their study. Dietary levels of 10 and 20 ppm did not result in any identifiable symptoms of parathion poisoning. Subsequent examination did not reveal any pathological lesions. A dietary level of 50 ppm was toxic and treatment at this level resulted in 42% mortality during the year, with most of the deaths occurring early in the test period when the animals were young. At dietary levels of 75 and 100 ppm most of the rats died during the first few weeks.

Chronic (dietary) toxicity of parathion fed to rats at several levels (10,25,50, and 100 ppm in diet) was studied by Hazelton and Holland (1950). Their tests were conducted for 104 weeks using male rats and for 64 weeks using female rats. With the male rats 40% mortality was observed for controls over the test period. Rats fed parathion in the diet at 50 ppm had a mortality level of 20% and those treated at a dietary level of 100 ppm had a mortality level of 38%. In the female rats a relatively high mortality (33%) occurred in the controls and relatively few rats were included in each treatment group. However, a dietary level of 10 ppm parathion did not cause any mortalities (100% survival), and at 50 ppm the mortality rate was only about equal to that of the untreated controls (38%). At 100 ppm, female rats exhibited evidence of toxicity and appeared unthrifty, indicating that chronic toxicity, like acute toxicity, is different for female and male rats.

All females in the control and 10-ppm treatment groups produced living litters. All but one female in the 50-ppm treatment group bore living litters.

Based on changes in cholinesterase levels, the "no-effect" level of parathion in the rat was reported by Edson et al.,(1964)[1/] to be 0.02 mg/kg/day when the insecticide was fed over an 84-day period. The smallest effect was found in the range 0.04 to 0.06 mg/kg/day. These studies indicated that in the rat a wide margin of safety existed between the maximum "no-effect" level over that needed to evoke a frank toxic effect.

Toxicity of parathion in combination with other drugs - The effect of central-depressant drugs on toxicity of parathion for rats has been

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[1/] Edson, E. F., et al., "Summaries of Toxicological Data: No-Effect Levels of Three Organophosphates in the Rat, Pig, and Man," Food Cosmet. Toxicol., 2:311 (1964).

Table 10. CHRONIC ORAL TOXICITY TEST IN RATS FED PARATHION[a]

Concentration of parathion in feed (ppm)	Duration of test (days)	Mortality due to poisoning (%)	Comments
100	19	80	First death occurred in 2 hr. All survivors had severe symptoms of poisoning.
75	27	73	Slight symptoms in 2 hr. After second week food consumption irregular.
50	365	42	First symptoms and first death on seventh day. Deaths occurred through third week but not after. Symptoms decreased in severity and frequency after third month.
20	365	0	Symptomless for entire test period.
10	365	0	Symptomless for entire test period.

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[a] Adapted from the data of Barnes and Denz, *op. cit.* (1951).

studied by Weiss and Orzel (1967).[1] Following treatment with reserpine, chlordiazepoxide, hexabarbital and phenobarbital, female rats were treated with 2, 4, or 8 mg/kg parathion. The greatest enhancement of toxicity was obtained at 2 and 4 mg/kg parathion. Alcohol was found to increase toxic effects of parathion only at the 4-mg/kg dose level.

The interactions (toxicity) between polychlorinated biphenyls (PCB) and parathion in rats was studied by Phillips et al. (1972).[2] The rationale behind the study was that because PCB ingestion can modify microsomal enzyme activity in rabbits and rats, it is possible that organophosphate toxicity may also be altered. Adult female rats were used in the tests. Plasma and brain cholinesterase activities were used to measure the effects of exposure to PCB, parathion and combinations of the two materials. The PCB was given orally (in diet) and parathion was injected intraperitoneally.

The results of these studies showed that the feeding of PCB (Aroclor 1221) had no effect on plasma cholinesterase. It was also found that parathion did not reduce the plasma or brain cholinesterase activity in animals fed PCB's below that observed in animals which were not fed PCB's. The conclusion drawn was that chronic ingestion of PCB (Aroclor 1221) up to a level of 200 ppm in the diet did not potentiate parathion toxicity. Potentiation of parathion toxicity did not result from feeding of Aroclor 1254 up to 20 ppm. The effect of Aroclor 1254 when fed at 200 ppm makes interpretation of parathion toxicity effects difficult.

Acute oral toxicity - mice - A summary of the data for acute oral toxicity of parathion to mice is given in Table 11. The average LD_{50} value for males was 23.0 mg/kg (range 17.5 to 30.3 mg/kg) and for male-female groups 12.7 mg/kg (range 6.0 to 25.0 mg/kg). It appears that the susceptibility to parathion poisoning for male rats is higher than for male mice.

In mice, as with most other species studied, there is no appreciable tissue accumulation of residuals, and deaths generally occur only in cases where parathion is administered as an acute toxic (lethal) dose. In general, prompt and apparently complete recovery from nonlethal doses

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[1] Weiss, L. R., and R. A. Orzel, "Enhancement of Toxicity of Anticholinesterases by Central Depressant Drugs in Rats," Toxicol. Appl. Pharmacol., 10:334-339 (1967).

[2] Phillips, W. E. J., G. Hatina, D. C. Villeneuve, and D. L. Grant, "Effect of Parathion Administration in Rats Following Long-Term Feeding with PCB's," Environ. Physiol. Biochem., 2:165-169 (1972).

Table 11. ACUTE ORAL TOXICITY OF PARATHION TO MICE

Acute oral LD_{50} (mg/kg)			
Male	Female	Mixed	Reference
-	-	8.1	a/
-	-	6.0	b/
-	-	25.0	c/
18.5	-	-	d/
-	-	21.0	b/
-	-	6.4	e/
-	-	16.9	e/
-	-	6.7	e/
-	-	11.6	e/
17.5	-	-	f/
25.6	-	-	g/
30.3	-	-	g/
23.0 Average		12.7 Average	
7.0-30.3 Range		6.0-25.0 Range	

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a/ DuBois, K. P., *op. cit.* (1971).

b/ Hazelton and Holland, *op. cit.* (1950).

c/ Frawley et al., *op. cit.* (1952).

d/ Vukovich, R. A., A. J. Triolo, and J. M. Coon, "The Effect of Chlorpromazine on the Toxicity and Biotransformation of Parathion in Mice," *J. Pharmacol. Exp. Thera.*, 178(2):395-401 (1971).

e/ Rosival, L., F. V. Selecky, and L. Vbrovsky, "Acute Experimental Poisoning with Organophosphorus Insecticides," *Bratisl. Lek. Listy*, **38**:151-160 (1958).

f/ Triolo, A. J., and J. M. Coon, "Toxicologic Interactions of Chlorinated Hydrocarbon and Organophosphate Insecticides," *J. Agr. Food Chem.*, 14(6):549-555 (1966).

g/ Konst, H., and P. J. Plummer, "Acute and Chronic Toxicity of Parathion to Warm-Blooded Animals," *Can. J. Comp. Med. Vet. Sci.*, **14**:90-108 (1950).

is observed with mice and other laboratory animals. Animals that survive a single dose longer than 24 hr may generally be expected to recover completely, and there is no apparent residual toxicity (Hazelton and Holland, 1950). However, because depressed blood cholinesterase lasts throughout the lifetime of red blood cells, this symptom remains until the red blood cells are naturally replaced.

Acute toxicity - mice, routes other than oral - The toxicity of parathion by intraperitoneal, intravenous and subcutaneous injection into mice is shown in Table 12. There appears to be close agreement in the LD_{50} values for male-female groups injected intraperitoneally and subcutaneously. There were not sufficient reports found in the literature on males and females alone to make a valid comparison of LD_{50} values.

Natoff (1967)[1/] reported mice to be more susceptible to parathion administered intravenously than by other routes. However, in a more recent study this same author indicated that the intraperitoneal route was more lethal. Parathion was shown to be more toxic to female mice by intraperitoneal injection than by intravenous injection. The estimated median lethal dose by oral route was 85.6 μM/kg, by intraperitoneal injection 50.3 μM/kg, by intravenous injection 58 μM/kg, and subcutaneously 71.3 μM/kg. These values average to about 68 μM/kg for the hepatic routes and 65 μM/kg for the peripheral routes (Natoff, 1970[2/]).

Subacute and chronic oral toxicity - mice - No information was found concerning either the subacute or the chronic oral toxicity of parathion to mice.

Acute oral toxicity - guinea pigs - The acute oral toxicity of parathion for guinea pigs as reported by various workers is given in Table 13. The LD_{50} value for guinea pigs, male-female groups, varied from 9.3 mg/kg to 32.0 mg/kg of body weight. The LD_{50} values for male guinea pigs were 16.3 mg/kg and 24.0 mg/kg of body weight.

[1/] Natoff, I. L., "Influence of the Route of Administration on the Toxicity of Some Cholinesterase Inhibitors," J. Pharm. Pharmacol., 19:612-616 (1967).

[2/] Natoff, I. L., "Influence of the Route of Exposure on the Acute Toxicity of Cholinesterase Inhibitors," Europ. J. Toxicol., 3:363-367 (1970).

Table 12. ACUTE TOXICITY OF PARATHION TO MICE--ROUTES OTHER THAN ORAL

Route[a]	LD$_{50}$ (mg/kg) Male	Female	Mixed	Reference
IP	10	10	-	b/
	13.5	-	-	c/
	-	14.7	-	d/
	-	-	8.2	e/
	-	-	14.4	e/
	-	-	4.6	e/
	-	-	8.8	e/
			9.0 Average	
			4.6-14.4 Range	
IV	7.6	-	-	f/
	-	16.9	-	d/
	-	-	5.4	e/
	-	-	1.0	e/
SC	-	20.8	-	d/
	-	-	11.6	e/
	-	-	13.6	e/
	-	-	7.8	e/
			11.0 Average	
			7.8-13.6 Range	

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a/ IP - Intraperitoneal; IV - Intravenous; SC - Subcutaneous.

b/ DuBois et al., op. cit. (1949).

c/ Benke, G. M., K. L. Cheever, F. E. Mirer, and S. D. Murphy, "Comparative Toxicity, Anticholinesterase Action and Metabolism of Methyl Parathion in Sunfish and Mice," Toxicol. Appl. Pharmacol., 28:97-109 (1974).

d/ Natoff, I. L., op. cit. (1967).

e/ Rosival et al., op. cit. (1958).

f/ Vukovich et al., op. cit. (1971).

Acute toxicity - guinea pigs, routes other than oral - The dermal LD_{50} for parathion in guinea pigs is reported as 0.60 ml/kg on intact skin and 0.80 ml/kg on abraded skin (Roudabush et al., 1965[1]). The LD_{50} for intraperitoneal injection is 12.0 mg/kg (Anon., Council of Europe, 1964[2]).

Subacute and chronic oral toxicity - guinea pigs - No subacute or chronic oral toxicity data for guinea pigs were found relative to parathion.

Acute, subacute and chronic toxicity - dogs - It has been reported (Anon., Council of Europe, 1964) that acute oral toxicity of parathion in dogs is 3.0 to 5.0 mg/kg. The intraperitoneal LD_{50} values for dogs is 3 to 5 mg/kg of body weight (DuBois et al., 1949); 12 to 20 mg/kg (Anon., Council of Europe, 1964). By intravenous injection, the lethal dose is 12 to 20 mg/kg (Anon., Council of Europe, 1964).

Table 13. ACUTE ORAL TOXICITY OF PARATHION TO GUINEA PIGS

Sex	LD_{50} (mg/kg)	Reference
Male and female	9.3	a/
Male and female	16.2	b/
Male and female	19.8	b/
Male and female	32.0	c/
Male	24.0	b/
Male	16.3	b/

a/ Hazelton and Holland, op. cit. (1950).
b/ Konst and Plummer, op. cit. (1950).
c/ Frawley et al., op. cit. (1952).

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[1] Roudabush, R. L., C. J. Terhaar, D. W. Fassett, and S. P. Dziuba, "Comparative Acute Effects of Some Chemicals on the Skin of Rabbits and Guinea Pigs," Toxicol. Appl. Pharmacol., 7:559-565 (1965).

[2] Anon., Council of Europe-Netherlands Report on Parathion and Parathion-Methyl, Ministerie von Sociale Zahen En Volksgezandheid (1964).

The subacute toxicity of parathion for dogs was studied by Frawley and Fuyat (1957)[1] by measuring changes in plasma and RBC cholinesterase. When parathion was incorporated in the dogs' diet at 1 ppm and fed for 24 weeks, a significant (minimal) reduction in plasma cholinesterase occurred. At 2 and 5 ppm plasma cholinesterase reductions of 60 and 70% were recorded. Inhibition of RBC cholinesterase occurred where diet levels of parathion were 2 ppm or above.

Chronic (dietary) toxicity of parathion for dogs was determined by Hazelton and Holland (1950) using 15% wettable powder given in gelatin capsules 6 days/week for 90 days. The capsules were given either with a meal or immediately afterward (prior administration resulted in loss of appetite and food refusal).

At 2 mg/kg/day the dog lived 3 weeks but exhibited toxic signs continuously. At 1 and 3 mg/kg/day the animals survived for the full 90-day test period. During the early stages the dogs were nervous and irritable but during the final month both dogs were normal in their behavior. Neither dog showed evidence of gross pathology. Histopathological examination revealed some degenerative changes in the liver.

Acute, subacute and chronic toxicity - cats - No data were found in the open literature on the acute, subacute and chronic oral toxicity of parathion to cats. The subcutaneous, intraperitoneal and intravenous LD_{50}'s are 15.0 mg/kg, 3.0 to 5.0 mg/kg and 3.0 to 5.0 mg/kg, respectively (Anon., Council of Europe, 1964). DuBois et al. (1949) reported an intraperitoneal LD_{50} value of 12 to 20 mg/kg of body weight.

In cats, parathion has been shown to be readily absorbed through the skin in unchanged form, although no dermal LD_{50} values have been reported (Fredriksson, 1964[2]).

Acute, subacute and chronic toxicity - rabbits - The acute oral LD_{50} for male rabbits is 68 mg/kg of body weight as reported by Konst and Plummer (1950).

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[1] Frawley, J. P., and H. N. Fuyat, "Effect of Low Dietary Levels of Parathion and Systox on Blood Cholinesterase of Dogs," J. Agr. Food Chem., 5:346 (1957).

[2] Fredriksson, T., "Studies on the Percutaneous Absorption of Parathion and Paraoxon. Part VI. In vivo Decomposition of Paraoxon During the Epidermal Passage," J. Invest. Dermatol., 42:37-40 (1964).

The acute dermal LD_{50} for mixed sexes of rabbits is 0.07 ml/kg (Roudabush et al., 1965). Diechmann et al (1952)[1] reported the dermal LD_{50} of parathion for rabbits is 870 mg/kg, which was similar to his findings on guinea pigs. Lehman (1948), on the other hand, reported the acute dermal LD_{50} for rabbits to be 40-50 mg/kg. In rabbits, it has been shown that parathion is absorbed dermally in an unchanged form (Fredriksson et al., 1961[2].

No information was found concerning subacute or chronic studies in rabbits.

Toxicity to Domestic Animals -

Goats - Of the ruminants which have been studied, the goat appears most susceptible to parathion poisoning. Although no LD_{50} values have been reported for this animal, daily subacute oral doses as low as 8 mg/kg were reported to have killed one experimental goat in 11 days. Other subacute doses reported for goats range from 12 mg/kg for 5 days to 32 mg/kg for 10 days (Wilber and Morrison, 1955[3]). Unlike other ruminant species studied, parathion is reported to cross the placental blood barrier and exert anticholinesterase effects on the fetus. Parathion has been reported to appear in the milk of lactating female goats. It is not known how long the anticholinesterase activity of milk from poisoned goats persists (Wilber and Morrison, 1955).

Sheep - The acute oral LD_{50} of parathion for ewe and ram lambs is estimated at 40 to 50 mg active ingredient per kilogram of body weight. Lethal doses for lambs range from four doses of 24 mg/kg each to three doses of 48 mg/kg each. The insecticide was not markedly cumulative in its toxic effects (Mol et al., 1972[4]). Sheep receiving daily subacute doses exhibit symptoms similar to those in acutely affected animals. Affected sheep that survived recovered completely after cessation of treatment, and appeared to be normal with no recurring ill effects.

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[1] Diechmann, W. B., W. Pugliese, and J. Cassidy, "Effects of Dimethyl and Diethyl Paranitrophenyl Thiophosphate on Experimental Animals," AMA Arch. Ind. Hyg. Occup. Med., 5:44-51 (1952).

[2] Fredriksson, T., W. L. Farrior, Jr., and R. F. Witter, "Studies on the Percutaneous Absorption of Parathion and Paraoxon. Part I. Hydrolysis and Metabolism Within the Skin," Acta Derm. Venerol., 41:335-343 (1961).

[3] Wilber, C. G., and R. A. Morrison, "The Physiological Action of Parathion in Goats," Am. J. Vet. Res., 10(59):308-313 (1955).

[4] Mol, J. C. M., D. L. Harrison, and R. H. Tlefer, "Parathion: Toxicity to Sheep and Persistence on Pasture and in Soil," N. Z. J. Agr. Res., 15:306-320 (1972).

The maximum safe oral dose (MSD) for sheep has been determined to be 10 mg/kg, whereas the minimum toxic dose (MTD) is 20 mg/kg (Radeleff et al., 1955[1]).

Data obtained from grazing studies (Mol et al., 1972) indicate that sheep may be grazed, with no ill effects, on pastures following heavy spraying with parathion. In this study pastures were sprayed with up to 6.0 lb/acre (approximately seven to 27 times normal application rate). Sheep were grazed on plots for 28-day periods beginning at 0 to 30 days after spraying. In the most severe case (28 days grazing immediately following spraying), sheep developed no symptoms of poisoning except slight scouring, even when their blood cholinesterase was almost completely inactivated.

Cattle - Dahm et al (1950)[2] fed two groups of lactating cattle 1 ppm and 5 ppm of parathion for 81 days. The parathion was given by capsule. These levels provided 0.022 and 0.112 mg/kg/day, and they produced no noticeable adverse effects. Two other cows were fed 5 ppm of parathion the first week. The amount was increased each week until 40 ppm was being consumed per week. The lowest and highest levels were equivalent to 0.11 and 0.89 mg/kg/day. No adverse effects were observed.

The MSD and MTD for 1- to 2-week old calves have been conservatively estimated to be 0.25 and 0.5 mg/kg, respectively, whereas the MSD for year-old cattle is 4.0 mg/kg (Radeleff et al., 1955). A more thorough study carried out by Pankaskie et al. (1952)[3] indicates that the above estimates may be low. Cattle were fed a ration containing baled alfalfa hay for a 61-day period. The average parathion concentration of all hay fed over the 61-day period was 13.6 ppm. During this period no parathion residues or degradation products could be detected in the urine, milk, or jugular blood of the test animals. In an additional experiment a cow was fed parathion by direct oral administration of daily initial doses of 1 mg/kg for 14 weeks. The dosage was increased weekly until the daily dose reached 32 mg/kg of body weight. The authors stated that this level was the highest intake by any survivors. During this time, no symptoms of parathion poisoning appeared. To rule out development of tolerance over the 14-week feeding period, another cow was fed 16 mg/kg/day for 1 week without adverse effects. As with the alfalfa feeding experiments, no residues of parathion or p-nitrophenol were detected in urine, jugular blood or milk. These experiments indicate that adult dairy cattle are less susceptible to parathion poisoning than other ruminants.

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[1] Radeleff, R. D., G. T. Woodard, W. J. Nickerson, and R. C. Bushland, "Part II. Organic Phosphorus Insecticides," *The Acute Toxicity of Chlorinated Hydrocarbon and Organic Phosphorus Insecticides to Livestock*, U.S. Department of Agriculture, Tech. Bull. 1122, pp. 35-46 (1955).

[2] Dahm, P. A., F. C. Fountaine, J. C. Pankaskie, R. C. Smith, and F. W. Atkeson, "The Effects of Feeding Parathion to Dairy Cows," *J. Dairy Sci.*, 33(101):747-757 (1950).

[3] Pankaskie, J. E., F. C. Fountaine, and P. A. Dahm, "The Degradation and Detoxification of Parathion in Dairy Cows," *J. Econ. Entomol.*, 45:51-60 (1952).

Radeleff and Bushland (1960)[1] in a symposium on "The Nature and Fate of Chemicals Applied to Soils, Plants and Animals" reiterated an observation made earlier in some of their toxicological studies: with toxic chemicals that were not of equal toxicity to adult and young animals the dairy calf is the most susceptible of all farm animals (Radeleff and Bushland, 1960).

The minimum toxic dose of parathion (used as a dip or spray) for 1- to 2-week old calves was reported as 0.01% while that for sheep and goats (adults) was 1.0%.

Additional evidence for the greater sensitivity of calves to parathion (compared to steers and sheep) was reported by Radeleff and Woodard (1957)[2] and Radeleff (1958).[3] A lethal dose of parathion for calves was reported to be 1.5 mg/kg while a lethal dose for steers and sheep was 75 and 20 mg/kg, respectively. Twenty-five milligrams per kilograms was reported to be nontoxic for steers, 10 mg/kg was nontoxic for sheep but the nontoxic dose for calves was 0.25 mg/kg.

Symptomatology and Pathology Associated with Mammals - While the symptoms may vary in intensity with dosage, mammals can be expected to exhibit those symptoms typical of cholinergic poisoning. Among these symptoms are muscle faciculation, excessive salivation, tremor, miosis, partial paralysis, labored breathing, lachrymation, diarrhea, involuntary urination, convulsion, depression and death (Hazelton and Holland, 1950; Gaines, 1960; Konst and Plummer, 1950).

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[1] Radeleff, R. D., and R. C. Bushland, "The Toxicity of Pesticides for Livestock," Symposium: The Nature and Fate of Chemicals Applied to Soils, Plants, and Animals," Agricultural Research Service, U.S. Department of Agriculture (27-29 April 1960).

[2] Radeleff, R. D., and G. T. Woodard, "The Toxicity of Organic Phosphorus Insecticides to Livestock," J. Am. Vet. Med. Assoc., 130: 215-216 (1957).

[3] Radeleff, R. D., "The Toxicity of Insecticides and Herbicides to Livestock," Adv. Vet. Sci., 4:265-276 (1958).

In acute poisonings autopsy findings usually reveal only gastrointestinal irritation and pulmonary hemorrhage.

In rats fed at relatively high dietary levels of parathion for long periods of time, the symptoms reported are those of acute poisoning (salivation, lachrymation, intestinal hyperperistalsis and muscular tremors) occurring as recurrent episodes.

Pathologically, chronically poisoned animals show the same lesions as those acutely poisoned--vacuolation and collapse of the acinar structure of the submaxillary gland and pancreas along with atrophy of the thymus and spleen (Barnes and Denz, 1951). These symptoms are most prominent during the early exposure period and may diminish with continued feeding.

Physiological and Pharmacological Aspects of Parathion - The physiology, pharmacology and toxicology of parathion poisoning have been described by Hamblin and Golz (1955).[1] These authors state that the only important pharmacological action of parathion is its inhibiting action on the enzyme acetylcholinesterase.

Acetylcholine is needed for nerve impulse transmission, and this need is met by the action of the enzyme choline acetylase. The hydrolysis of acetylcholine to choline and acetic acid is accomplished by another enzyme, acetylcholinesterase. There are two types of cholinesterases, true and pseudocholinesterases. The former is found in nerve and muscle tissue and in the erythrocytes. Pseudocholinesterase is actually a group of enzymes which are found in the pancreas and salivary glands and other tissues. The latter enzymes hydrolyze acetylcholine more slowly than acetylcholinesterase.

The plasma cholinesterase in most species, including man, is pseudocholinesterase. The concentration varies widely from individual to individual, and human erythrocytes contain true cholinesterase.

The absorption of parathion brings about the accumulation of acetylcholine, because of a failure in the disposal mechanism in parasympathetic post-ganglionic fibers, in sympathetic ganglia and in the central nervous system as well as at the myoneural junction of muscle.

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[1] Hamblin, D. O., and H. H. Golz, "Parathion Poisoning, A Brief Review," Ind. Med. Surg., 24(22):65-72 (1955).

Autopsy examination of animals succumbing to lethal dosages of parathion, in either single or multiple doses, has not revealed extensive or wide-spread tissue damage that could be related to treatment with the compound.

The only accumulative aspects of parathion are suggested by the acetylcholine recovery period being contingent upon the death of the red blood cell. Furthermore, the inhibition of the enzyme is not reversible. Repeated doses of parathion will produce steadily decreasing levels of enzyme activity in the blood.

In the diagnosis of parathion poisoning, the RBC cholinesterase level is one of the more important clinical measurements. Plasma cholinesterase is nonspecific and it has no functional relationship with the activity of the nervous system. Exposure to anti CHE agents is assumed when the plasma and RBC cholinesterase activity is depressed by 25%. The excretion of p-nitrophend in the urine confirms this assumption.

In acute poisoning, manifestations usually occur only after more than 50% of the plasma cholinesterase is inhibited. After an acute poisoning, it takes about 4 weeks for plasma cholinesterase to return to normal.

Summary - Parathion is a highly toxic pesticide, as evidenced by studies with laboratory and domestic animals, fish, other aquatic life, avian species, wildlife and humans. The oral LD_{50} value for rats is about 7 mg/kg (range 2 to 30 mg/kg) for males and 4.0 mg/kg (range 2 to 6 mg/kg) for females. The LC_{50} value for inhalation toxicity has been reported as 0.0315 mg/liter (4-hr exposure) to 0.115 mg/liter (1-hr exposure). The LD_{50} value for intraperitoneal injection ranges from 3.6 to 7.0 mg/kg. Dermal toxicity is much less pronounced than the other toxicity measurements, ranging from 6.8 to 200 mg/kg; most of the reported results were above 20 mg/kg.

No mortality occurred when rats were fed parathion for 15 to 16 weeks at 0.52 mg/kg/day or at 2.4 mg/kg/day. The mortality went up to 30% when the average daily dosage was increased to 15.4 mg/kg. Over a 1-year period the mortality was 42% for rats fed 50 ppm of parathion. The mortality rose to 80% over a 19-day feeding period with a concentration of parathion in the feed equal to 100 ppm.

Mice are not as susceptible to parathion poisoning as rats. The average LD_{50} for males was 19.8 mg/kg (range 7.0 to 30.3 mg/kg) and for male-female groups, 12.7 mg/kg (range 6.0 to 25.0 mg/kg).

The intraperitoneal and intravenous LD_{50} values for mice were not as low as for rats. In general, the order of toxicity for mice was about the same for the intraperitoneal and intravenous routes. Subcutaneous injections yielded high LD_{50} values (7.8 to 21.4 mg/kg).

No information was found on long-term feeding studies with mice. Only one paper reported a dermal toxicity of 0.60 ml/kg (intact skin) and 0.80 ml/kg (abraded skin).

The oral toxicity of parathion for guinea pigs was close to mice (LD_{50} range 9.3 mg/kg to 32.0 mg/kg). The LD_{50} value for intraperitoneal injection was 12.0 mg/kg.

No information was found for the subacute and chronic oral toxicity of parathion to guinea pigs.

The information on the toxicity of parathion in dogs was sparse. One report on acute oral toxicity indicated that 3.0 to 5.0 mg/kg was lethal. Two reports on the intraperitoneal LD_{50} value were at variance--12 to 20 mg/kg and 3 to 5 mg/kg.

The subcutaneous, intraperitoneal and intravenous LD_{50} values are 15.0 mg/kg, 3.0 to 5.0 mg/kg and 3.0 to 5.0 mg/kg, respectively, for cats. Another report indicated that the intraperitoneal LD_{50} for parathion in cats was 12 to 20 mg/kg. No dermal toxicity data is available. No information was found on subacute and chronic oral toxicity to cats.

One report was found for the LD_{50} (68 mg/kg) for oral ingestion by rabbits. The acute dermal LD_{50} appears to be about 0.07 mg/kg (abraded skin).

There was one report on the toxicity of parathion to goats. No LD_{50} values were reported. Daily doses of 8 mg/kg killed one animal in 11 days.

The maximum safe oral dose for sheep has been determined to be 10 mg/kg and the minimum toxic dose as 20 mg/kg. One investigator reported that the acute oral LD_{50} of parathion for lambs was estimated to be 45 to 50 mg/kg.

Parathion has been fed to cattle at 1 ppm and 5 ppm/day for 81 days without any noticeable effect. One dairy cow has been fed 32 mg/kg/day for 7 days without adverse effects. The same animal had been fed for a number of weeks at lower gradually increasing levels.

These collective data indicate that parathion is highly toxic to all species tested thus far by all routes. There is usually a lesser degree of toxicity produced by dermal exposure than by other routes. The vehicle is very important in dermal toxicity tests. Episodes of acute toxicity are more likely to occur in nature than are chronic poisonings. The pathology of chronic toxicity is similar to that found in acute episodes. The young are more susceptible than the mature organisms. There is a unique sex aspect in some species (male rats are less susceptible than females, whereas in most other species sex differences are not as great). The symptoms and mechanisms of physiological action in all species are essentially the same. Cholinesterase depression is a sensitive clinical measurement and in some instances too sensitive (chronic exposures). The pathological damage appears to be minimal even in chronic exposures.

Metabolism of Parathion

The metabolism of parathion is discussed in the following seven subsections: absorption, distribution, excretion, biotransformation, degradation, tissue residues, and summary.

Absorption - Parathion is absorbed in animals by the oral, inhalation or dermal routes. Nabb et al. (1966)[1] have shown that parathion was absorbed through rabbit skin at the rate of 0.059 ug/min/cm^2 based on the inhibition of red cell acetylcholinesterase activity. Fredriksson (1964) measured the rate of absorption of ^{32}p-paraoxon through cat skin. When 50 ul of paraoxon was applied to 4.1 cm^2 of skin the absorption rate was found to be about 6 muM/min/cm^2. Blood levels 3 hr later were 0.05 ug of paraoxon per milliliter. However, approximately 90% of the paraoxon was detoxified during epidermal passage due to a skin enxyme in cats that can split the paraoxon molecule. In a previous study, Fredrikson (1961b) also reported[2] the rate of paraoxon absorption to be 0.47 to 0.95 muM/min/cm^2 through cat skin.

Parathion was 10 times less toxic than paraoxon when given intravenously but was 55 times less toxic when applied to the skin of rabbits (Nabb et al., 1966).

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[1] Nabb, D. P., W. J. Stein, and W. J. Hayes, Jr., "Rate of Skin Absorption of Parathion and Paraoxon," Arch. Environ. Health, 12:501-505 (1966).

[2] Fredriksson, T., "Studies on the Percutaneous Absorption of Parathion and Paraoxon. Part III. Rate of Absorption of Parathion," Acta Derm. Venerol., 41:353-363 (1961b).

A comparative study of the acute dermal toxicity and primary irritation in rabbits and guinea pigs indicated that parathion was more toxic to rabbits (Roudabush et al., 1965).

Distribution - The dermal and intravenous administration of ^{35}S-parathion to rabbits showed very little accumulation of ^{35}S in the blood, organs or tissues (dermal 10 days after exposure; intravenous 1 hr after exposure) (Jensen et al., 1952[1/]). Feeding 167 mg of parathion in baled hay to cows did not produce detectable levels of parathion or *p*-nitrophenol in milk or blood (Pankaskie et al., 1952). In the same study cows were fed 1 to 32 mg/kg/day of parathion by capsule, and did not produce detectable levels of parathion, *p*-nitrophenol or *p*-aminophenol in the milk or blood. Giachetti et al. (1966)[2/] measured the amount of paraoxon in rat brain after treatment with 1 mg/kg of parathion by intraperitoneal injection. At 6 hr after treatment there was 30 to 33 ng of paraoxon, 2 to 3 ng after 18 hr and only a trace at the end of 72 hr. Taylor and Gaut (1968)[3/] have written a review describing the distribution and binding of parathion and paraoxon at the enzyme level in many tissues.

Excretion - Cows fed parathion excreted aminoparathion, diethylphosphoric acid, and phosphorothioic acid in the urine and about 1% of the dose in the milk (Ahmed et al., 1958[4/]). Cows probably also hydrolyze parathion to *p*-aminophenol and excrete *p*-aminophenol glucuronide (Pankaskie et al., 1952; Ahmed et al., 1958). Andersen and Karlog (1963)[5/] found from *in vitro* experiments with fluid that 75 to 90% of the parathion is reduced to aminoparathion or intermediates after 10 min; conjugated *p*-aminophenol was the dominant metabolite found in the urine. Cook (1957)[6/] had reported earlier that the reduction of parathion to *p*-aminoparathion was almost complete in 1 hr.

[1/] Jensen, J. A., W. F. Durham, and G. W. Pearce, "Studies on Fate of Parathion in Rabbits, Using Radioactive Isotope Techniques," *AMA Arch. Ind. Hyg. Occup. Med.*, 6:326-331 (1952).

[2/] Giachetti, A., C. Grasso, and G. Bernardi, "Persistence of O,O-Diethyl-O-para-nitrophenyl phosphate (Paraoxon) in the Brain of the White Rat Treated with a Single, Subtoxic Dose of Parathion," *Ric. Sci.*, 36(10):1077 (1966).

[3/] Taylor, W. J., and Z. N. Gaut, "Poisoning with the Newer Organophosphorus Insecticides," *Int. J. Clin. Pharmacol.*, 1(3):175-183 (1968).

[4/] Ahmed, M. K., J. E. Casida, and R. E. Nichols, "Bovine Metabolism of Organophosphorus Insecticides: Significance of Rumen Fluid with Particular Reference to Parathion," *J. Agr. Food Chem.*, 6:740-746 (1958).

[5/] Andersen, A. A., and O. Karlog, "Elimination of Parathion in Cows After Oral and Dermal Administration," *Acta Vet. Scand.*, 4:156-169 (1963).

[6/] Cook, J. W., "*In vitro* Destruction of Home Organophosphates Pesticides by Bovine Rumen Fluid," *J. Agr. Food Chem.*, 5(11):859-863 (1957).

Rats treated with ^{35}S-parathion excreted ^{35}S-sulfate in the urine (Nakatsugawa et al., 1969[1]). Paranitrophenol has been shown to be excreted in the urine of monkeys (Lieben et al., 1952[2]), and man (Wolfe et al., 1970;[3] Roan et al., 1969[4]) following exposure to parathion.

Biotransformation - The biotransformation of parathion involves two distinct phenomena, activation and deactivation (detoxification).

Gage (1953)[5] found paraoxon in a liver extract of parathion-treated rats. Aldridge (1953)[6] found that the serum cholinesterase enzymes of rabbit, rat and horse have different substrate specificities and demonstrated an enzyme which hydrolyzed parathion. Ecobichon et al. (1963)[7] reported that human liver esterases were inhibited by compounds, including parathion, which require activation. Gaines et al. (1966)[8] found that rats infused with 1.41 mg/kg of parathion via the intestinal vein died. However, if 1.69 mg/kg of parathion was infused via the femoral vein, none died within 65 min. Thus, it seemed that passage through the liver increased parathion toxicity. Shishido and Fukami (1963)[9] reported that liver and kidney slices converted parathion to paraoxon. They also reported that the microsomal fraction and the microsomal supernatant solutions were active in this conversion. Both fractions required diphosphopyridine nucleotide (NAD) as a cofactor for this conversion.

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[1] Nakatsugawa, T., N. M. Tolaman, and P. A. Dahm, "Degradation of Parathion in the Rat," Biochem. Pharmacol., 18(5):1103-1114 (1969).

[2] Lieben, J., R. K. Waldman, and L. Krause, "Urinary Excretion of Paranitrophenol Following Parathion Exposure," AMA Arch. Ind. Hyg. Occup. Med., 6:491-495 (1952).

[3] Wolfe, H. R., W. F. Durham, and J. F. Armstrong, "Urinary Excretion on Insecticide Metabolites," Arch. Environ. Health, 21:711-716 (1970).

[4] Roan, C. C., D. P. Morgan, N. Cook, and E. H. Paschal, "Blood Cholinesterases, Serum Parathion Concentrates and Urine p-Nitrophenol Concentrations in Exposed Individuals," Bull. Environ. Contam. Toxicol., 4(6):362-369 (1969).

[5] Gage, J. C., "A Cholinesterase Inhibitor Derived from O,O-Diethyl O-p-Nitrophenyl Thiophosphate in vivo," Biochem. J., 54:426-430 (1953).

[6] Aldridge, W. N., "Serum Esterases, 2. An Enzyme Hydrolysing Diethyl p-Nitrophenyl Phosphate (E600) and Its Identity With the A-Esterase of Mammalian Sera," Biochem. J., 53:117-124 (1953).

[7] Ecobichon, D. J., and W. Kalow, "Action of Organophosphorus Compounds Upon Esterases of Human Liver," Can. J. Biochem. Physiol., 41:1537-1546 (1963).

[8] Gaines, T. B., W. J. Hayes, Jr., and R. E. Linder, "Liver Metabolism of Anticholinesterase Compounds in Live Rats: Relation to Toxicity," Nature, 209(5018):88-89 (1966).

[9] Shishido, T., and J. Fukami, "Studies on the Selective Toxicities of Organic Phosphorus Insecticides (II)," Botyu-Kagaku, 28(1):69-76 (February 1963).

Nakatsugawa and Dahm (1967)[1] used ^{35}S-parathion to demonstrate that ^{35}S was bound to liver microsomes (probably by a desulfuration reaction). Activation by microsomes required $NADPH_2$ and O_2. The reaction was inhibited by SKF-525A and other synergists.

Nevkovic et al. (1973)[2] demonstrated that both $NADH_2$- and $NADPH_2$-linked electron transport components were involved in the oxidative metabolism of parathion and that the activity of $NADH_2$ - cytochrome c reductase was significantly higher than $NADPH_2$ - cytochrome c reductase.

Orzel and Weiss (1966)[3] used rat brain acetylcholinesterase inhibition as a measure of the conversion of parathion to paraoxon. Enzyme inhibition was decreased by oral administration of γ-aminobutyric acid, but not by intrathecal administration. Inhibition of enzyme activity was increased by pretreatment with chloroform and isonicotinic hydrazide, a monoamine oxidase inhibitor. Pretreatment with SKF-525A (a microsome inhibitor), arsenite (an oxidative enzyme inhibitor) or pyrogallol (a transferase inhibitor) did not alter the enzyme inhibition. Hitchcock and Murphy (1967a)[4] studied parathion metabolism in liver and kidney homogenates from rats and chickens. Kidney homogenates were half as active as liver homogenates based on the conversion of parathion to paraoxon. Liver required NADP and glucose-6-phosphate for maximal activity.

In 1969, Nakatsugawa et al. (1969) confirmed the initial in vivo conversion of parathion to paraoxon in male rats using ^{35}S and ^{32}P.

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[1] Nakatsugawa, T., and P. A. Dahm, "Microsomal Metabolism of Parathion," Biochem. Pharmacol., 16(1):25-38 (1967).

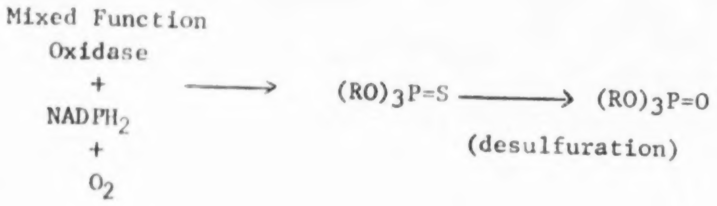
[2] Nevkovic, N., S. Vitorovic, and M. Plesnicar, "The Role of Liver Microsomal Enzymes in the Metabolism of Parathion," Biochem. Pharmacol., 22:2943-2946 (1973).

[3] Orzel, R. A., and L. R. Weiss, "The Effect of Various Chemicals on Rat Brain Cholinesterase Inhibition by Parathion," Arch. Int. Pharmacodyn. Thér., 164(1):150-157 (1966).

[4] Hitchcock, M., and S. D. Murphy, "Activation of Parathion and Guthion by Tissues of Mammalian, Avian, and Piscine Species," Fed. Proc., 26(2):427 (1967a).

Davison (1955)[1] found that the *in vitro* conversion of parathion was inhibited 52% by 10^{-3} M SKF-525A; 100% by iodoacetate, chloropicrin and 2,4-dinitrophenol; 90% inhibited by $HgCl_2$, hydroxylamine and choline; 66% inhibited by *p*-chloromercuribenzoate and not inhibited by cyanide. He also found that NAD and magnesium ions were required cofactors. Cytochrome c was not necessary for metabolism.

Fukuto and Metcalf (1969)[2] reviewed eight general reactions of the mixed function oxidate system, including the desulfuration of the phosphorothionates. One of the reactions is primarily associated with the conversion of parathion to paraoxon:



$$\begin{matrix}\text{Mixed Function} \\ \text{Oxidase} \\ + \\ \text{NADPH}_2 \\ + \\ O_2\end{matrix} \longrightarrow (RO)_3P{=}S \xrightarrow[\text{(desulfuration)}]{} (RO)_3P{=}O$$

Degradation of Parathion (and Paraoxon) - In 1958 Ahmed et al. demonstrated that rumen fluid from cows converted parathion to aminoparathion, and suggested that it was excreted as a glucuronide conjugate. Later, Williams (1970)[3] measured β-glucuronidase in rats after daily doses of 0.25, 0.5 and 1.0 mg/kg parathion for 4, 10 or 15 days. In females the serum β-glucuronidase was elevated in all cases. In males the β-glucuronidase was elevated only after 15 days of treatment. There was a decrease in serum β-glucuronidase in both sexes which was not dose-related.

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[1] Davison, A. N., "The Conversion of Schradan (OMPA) and Parathion into Inhibitors of Cholinesterase by Mammalian Liver," *Biochem. J.*, 61:203-209 (1955).

[2] Fukuto, T. R., and R. L. Metcalf, "Metabolism of Insecticides in Plants and Animals," *Ann. N.Y. Acad. Sci.*, 160:97-113 (1969).

[3] Williams, C. H., "β-Glucuronidase Activity in the Serum and Liver of Rats Treated with Parathion," *Toxicol. Appl. Pharmacol.*, 16:533-539 (1970).

Matsumura and Ward (1966)[1] found that human liver degraded parathion slightly faster than rat liver. Hitchcock and Murphy (1967b)[2] measured nitroreductase activity using parathion and paraoxon as substrates. When NADPH and FAD were used as cofactors, the mitochondria, microsomes and soluble fraction of rat liver contained 34, 38, and 28% of the nitroreductase activity.

In 1967 Neal (1967a)[3] used ^{32}p-labeled parathion to identify metabolites in the tissues of male and female rats, male mice and male guinea pigs. The metabolites found were paraoxon, diethyl hydrogen phosphate, diethyl hydrogen phosphorothionate and *p*-nitrophenol. Oxygen and NADPH were required for maximal activity. Phenobarbital or 3,4-benzopyrene pretreatment increased the activity by 65 to 130%. The reaction was inhibited by nitrogen and pure oxygen atmospheres as well as carbon monoxide. Continuing these studies, Neal (1967b)[4] concluded that (*in vitro*) inhibition of parathion metabolism indicated that the mixed function oxidase system was a degradative pathway. Inhibition with p-chloromercuribenzoate, cupric ion and 8-hydroxyquinoline was more effective against the conversion of parathion to paraoxon than against its conversion to diethyl hydrogen phosphorothionate. Conversion of parathion to diethyl hydrogen phosphorothionate was stimulated by EDTA, barium and calcium ions. The electron acceptors, flavin adenine dinucleotide (FAD), riboflavine, menadione and methylene blue exhibited a concentration-dependent inhibition of the conversion of parathion to paraoxon or diethyl hydrogen phosphorothionate. However, the inhibition constants were different.

A summary of these reactions is indicated by the following scheme:

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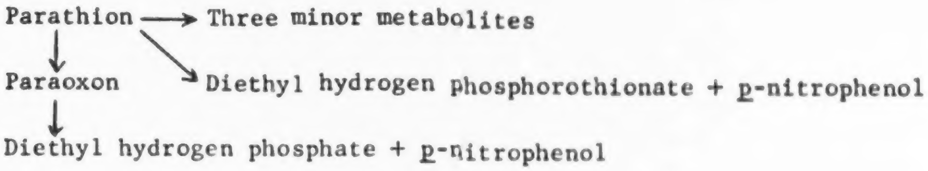
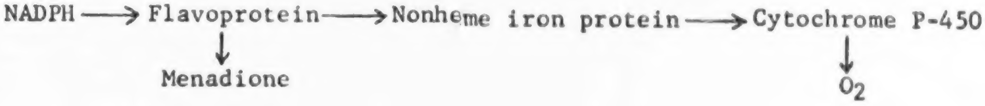
[1] Matsumura, F., and C. T. Ward, "Degradation of Insecticides by the Human and the Rat Liver," *Arch. Environ. Health*, 13:257-261 (1966).

[2] Hitchcock, M., and S. D. Murphy, "Enzymatic Reduction of O,O-(4-Nitrophenyl) Phosphorothioate, O,O-Diethyl O-(4-Nitrophenyl) Phosphate, and O-Ethyl O-(4-Nitrophenyl).Benzene Thiophosphonate by Tissues from Mammals, Birds, and Fishes," *Biochem. Pharmacol.*, 16(9):1801-1811 (1967b).

[3] Neal, R. A., "Studies of the Enzymic Mechanism of the Metabolism of Diethyl 4-Nitrophenyl Phosphorothionate (Parathion) by Rat Liver Microsomes," *Biochem. J.*, 105:289-297. (1967a).

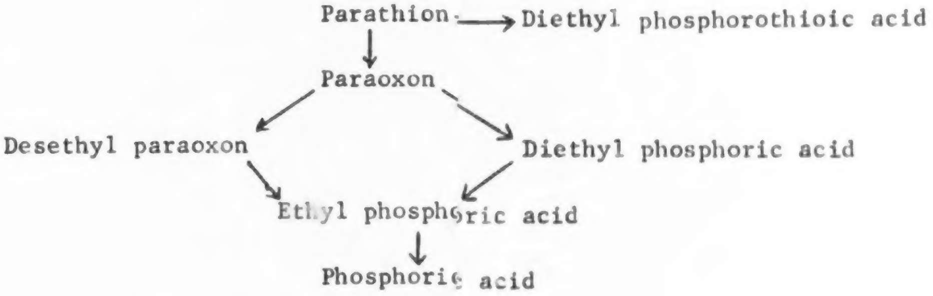
[4] Neal, R. A., "Studies on the Metabolism of Diethyl 4-Nitrophenyl Phosphorothionate (Parathion) *in vitro*," *Biochem. J.*, 103:183-191 (1967b).


Parathion
Three minor metabolites
Paraoxon
Diethyl hydrogen phosphorothionate + p-nitrophenol
Diethyl hydrogen phosphate + p-nitrophenol


NADPH
Flavoprotein
Nonheme iron protein
Cytochrome P-450
Menadione
O_2

Differences in inhibition constants and inhibitions suggest that different enzymes or different enzymes systems may be involved in the complete metabolism of parathion.

Studies by Nakatsugawa and Dahm (1967) showed that microsomes split the aryl phosphate bond, and that $NADPH_2$ and O_2 are required for the reaction. Further studies by Nakatsugawa et al. (1969) confirmed that aryl phosphate cleavage was mostly by liver microsomal oxidases, in vivo, using ^{32}P- and ^{35}S-labeled parathion. A minor portion was also catalyzed by nonoxidative soluble enzymes requiring reduced glutathione. They suggested the following metabolic pathway.


Parathion
Diethyl phosphorothioic acid
Paraoxon
Desethyl paraoxon
Diethyl phosphoric acid
Ethyl phosphoric acid
Phosphoric acid

In a review of parathion metabolism, Dahm (1970)[1/] suggested that paraoxon was not degraded by the same pathway as parathion. Paraoxon was metabolized by microsomes which required $NADPH_2$, nicotinamide, magnesium ion and potassium chloride for maximal activities.

[1/] Dahm, P. A., "Some Aspects of the Metabolism of Parathion and Diazinon," in Biochemical Toxicology of Insecticides, P. D. O'Brien and I. Yamamoto (Eds.), New York, Academic Press (1970).

Jacobsen et al. (1973) found that the parathion LD_{50} in rats did not change after partial hepatectomy and suggested that parathion toxicity is not due to the hepatic conversion of parathion to paraoxon. An alternate explanation for the results is that the remaining liver was sufficient to effect the conversion of parathion to paraoxon.

Bass et al. (1972)[1] reported that pretreatment of mice with DDT or DDE protected against parathion toxicity but not that of paraoxon. *In vitro*, the microsomes from mice given DDT or DDE converted more =S to =O than those from the controls. Detoxification of paraoxon was not altered by DDT or DDE. The increased rate of conversion of parathion to diethylphosphorothioic acid could explain the decreased parathion toxicity after DDT or DDE.

Alary and Brodeur (1970) found a close relationship between the LD_{50} of parathion and paraoxon in male and female rats and the *in vitro* liver oxidative and hydrolytic metabolism. Gagne and Brodeur (1972)[2] used equitoxic doses of ^{32}P-parathion in weanling and adult rats, and found that parathion was more toxic to weanlings mainly because of a lower liver enzyme activity. Brain tissue of weanlings seemed more susceptible than that of adults but not because of an increased facilitation of parathion or paraoxon across the blood-brain barrier.

Lichtenstein et al. (1973)[3] reported that soluble liver fractions (from 105,000 and 500,000 x g) degraded parathion (20%) and paraoxon (65%) to water-soluble metabolites which were nontoxic to insects.

Sakai and Matsumura (1971)[4] identified 14 esterase bands from human brain by thin-layer gel electrophoresis. Four of these were B-type esterases which degrade alkoxyphosphate sites, and six were A-type esterases which hydrolyze carboxyester sites.

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[1] Bass, S. W., A. J. Triolo, and J. M. Coon, "Effect of DDT on the Toxicity and Metabolism of Parathion in Mice," *Toxicol. Appl. Pharmacol.*, 22:684-693 (1972).

[2] Gagne, J., and J. Brodeur, "Metabolic Studies on the Mechanisms of Increased Susceptibility of Weanling Rats to Parathion," *Can. J. Physiol. Pharmacol.*, 50:902-915 (1972).

[3] Lichtenstein, E. P., T. W. Fuhremann, A. A. Hochberg, R. N. Zahlten, and F. W. Stratman, "Metabolism of [^{14}C] Parathion and [^{14}C] Paraoxon with Fractions and Subfractions of Rat Liver Cells," *J. Agr. Food Chem.*, 21(3):416-423 (1973).

[4] Sakai, K., and F. Matsumura, "Degradation of Certain Organophosphate and Carbamate Insecticides by Human Brain Esterases," *Toxicol. Appl. Pharmacol.*, 19(4):660-666 (1971).

Norman et al. (1973a)[1] reported that phenobarbital pretreatment in rats decreased the apparent activation energy of the mixed-function oxidase reactions. These results were not entirely due to an increase in cytochrome P-450.

Neal (1972)[2] measured the K_m for paraoxon and diethyl phosphorothioic acid in rabbit lung and liver and found lung to be much lower in activity. The V_{max} for lung microsomes was 20% of microsomes from liver. The mixed function oxidase from liver was induced by both phenobarbital and 3-methylcholanthrene, whereas only phenobarbital induced the lung mixed-function oxidase. In these studies, lung was about 3% as active as liver.

Norman et al. (1973b)[3] found that 3-methylcholanthrene and phenobarbital increased the metabolism rate of parathion by rough-surfaced, but not by smooth-surfaced microsomes, based on microsomal protein content. Phenobarbital and 3-methylcholanthrene increased the cytochrome P-450 content; there was no difference in the metabolic rate between the rough and smooth microsomes of untreated, and 3-methylcholanthrene- or phenobarbital-treated animals. However, based on the cytochrome P-450 in the entire microsome fraction, the metabolic rate was less in the 3-methylcholanthrene- and phenobarbital-treated rats than the controls. Cytochrome P-450 is the terminal enzyme of the mixed-function oxidase system. The iron in the heme moiety exists in both the hi- and low-spin state. There was no correlation between the spin state and the rate of parathion metabolism.

Villeneuve et al. (1970)[4] reported that SKF-525A had no effect on parathion toxicity in rats while DDT, benzpyrene and phenobarbital decreased the toxicity by a factor of 3. Phenobarbital and DDT decreased hexabarbital sleeping time, but benzpyrene and SKF-525A increased the sleeping time.

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[1] Norman, B. J., J. A. Roth, and R. A. Neal, "Effect of Temperature on the Mixed Function Oxidase-Catalyzed Metabolism of O,O-Diethyl p-Nitrophenyl Phosphorothionate (Parathion)," Toxicol. Appl. Pharmacol., 26:203-208 (1973a).

[2] Neal, R. A., "A Comparison of the in vitro Metabolism of Parathion in the Lung and Liver of the Rabbit," Toxicol. Appl. Pharmacol., 23:123-130 (1972).

[3] Norman, B. J., W. K. Vaughn, and R. A. Neal, "Studies of the Mechanisms of Metabolism of Diethyl p-Nitrophenyl Phosphorothionate (Parathion) by Rabbit Liver Microsomes," Biochem. Pharmacol., 22:1091-1101 (1973b).

[4] Villeneuve, D. C., W. E. J. Phillips, and J. Syrotiuk, "Modification of Microsomal Enzyme Activity and Parathion Toxicity in Rats," Bull. Environ. Contam. Toxicol., 5(2):125-132 (1970).

Hollingworth et al. (1973)[1] reported that glutathione-S-aryl transferase, a soluble enzyme, converted parathion to S-p-nitrophenylglutathione and diethylphosphorothioic acid. This enzyme can be differentiated from the glutathione-S-alkyl transferase that is also present in rat tissues.

Baeza et al. (1972)[2] injected rats with 10 mg/kg of parathion to produce a 50% inhibition of red cell acetylcholinesterase. This could be prevented by exposing the animals to 1,000 ppm of CO for 90 min prior to treatment. When 1.06 mg/kg of paraoxon was injected intraperitoneally no symptoms or mortalities were observed in rats. When the dose was increased to 1.42 mg/kg, symptoms were observed but there were no deaths. However, if 1,000 ppm of CO were given prior to 1.06 mg/kg of paraoxon, 80% of the animals exhibited symptoms and 40% died. If 2,500 ppm CO were given prior to administration of 1.42 mg/kg paraoxon, 70% of the animals died.

Parathion pretreatment decreased the normal metabolism of testosterone by rat liver microsomes (Stevens, 1973[3]). All polar and nonpolar metabolites were decreased except for a 30% increase in one dione.

Weiss et al. (1964)[4] reported that parathion did not alter the blood sugar levels in rabbits, dogs, or rats.

Taylor and Gaut (1968) reported that phenothiazines potentiated the toxicity of parathion.

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[1] Hollingworth, R. M., R. L. Alstott, and R. D. Litzenberg, "Glutathion S-Aryl Transferase in the Metabolism of Parathion and Its Analogs," Life Sci., 13:191-199 (1973).

[2] Baeza, C., A. M. Goldberg, and R. J. Rubin, "Effect of Carbon Monoxide on Response to Parathion and Paraoxon," Toxicol. Appl. Pharmacol., 22(2):288 (1972).

[3] Stevens, J. T., "The Effect of Parathion on the Metabolism of ^{3}H-Testosterone by Hepatic Microsomal Enzymes from the Male Mouse," Pharmacology, 10(4):220-225 (1973).

[4] Weiss, L. R., J. Bryant, and O. G. Fitzhugh, "Blood Sugar Levels Following Acute Poisoning with Parathion and 1-Naphthyl N-Methyl Carbamate (Sevin) in Three Species," Toxicol. Appl. Pharmacol., 6(3):363 (1964).

Tissue Residues - Jensen et al. (1952) found that intravenous ^{35}S-parathion did not accumulate in the blood, organs or tissues of rabbits. Pankaskie et al. (1952) found no accumulation of parathion or free p-aminophenol in the blood, milk or urine of cows fed 1 to 32 mg/kg/per day parathion. Roan et al. (1969) measured parathion levels in the serum of aerial applicator pilots. Some pilots had high serum parathion levels and low cholinesterase activity but were asymptomatic. Giachetti et al. (1966) gave rats 1 mg/kg of parathion IP and measured brain levels of paraoxon. At 6 hr after injection, the brain contained 30 to 33 ng of paraoxon. Only traces remained after 72 hr.

Summary - The following major points can be stated regarding the metabolism of parathion:

1. Parathion is readily absorbed through the skin, from the stomach and the lung.
2. Parathion is widely distributed throughout the body but does not accumulate at any site and causes no effect other than the irreversible binding of paraoxon to cholinesterase.
3. The major metabolites of parathion that are excreted are aminoparathion, p-nitrophenol, diethylphosphorothioic acid, ethylphosphoric acid and sulfate.
4. Metabolism of parathion is in two general steps: activation and detoxification.
5. Parathion is enzymatically hydrolyzed to paraoxon by the mixed-function oxidase system.
6. Oxidation of parathion and/or paraoxon is inhibited by carbon monoxide and SKF-525A.
7. Phenobarbital, 3-methylcholanthrene and benzpyrene stimulate parathion metabolism.
8. Microsomal oxidation of parathion requires $NADPH_2$, oxygen, potassium chloride and magnesium ion for maximum activity.

Effects on Reproduction

The effects of parathion on reproduction are reviewed in the following two subsections on domestic animals and wild avian species.

Domestic Animals - Some years ago, a study was made on the effect of parathion on reproduction and milk production in goats by Wilber and Morrison (1955). As a test material, they utilized parathion liquid with a purity of 98.76% or as a 25% wettable powder. They administered oral daily doses of 16 mg/kg (25% wettable powder) for 16 days. The total dosage given to one goat was 8,960 mg. After death the goat was found to be carrying two fetuses which would have been born in approximately 10 days. The authors pointed out that parathion did not induce an abortion in the goat. When another goat was killed near the terminal point of chronic poisoning, she was found to be carrying two 3-month-old fetuses. Superficially, the fetuses appeared normal in all respects. They found that the RBC cholinesterase activity (ΔpH) of heart blood of the fetuses was 0.14 and 0.17 respectively. The control value for the mother was about 1.05. However, her value dropped to 0% of normal before necropsy. In order to ascertain whether some anticholinesterase material crossed the placenta and exerted an effect on the fetus, fetuses were taken from unpoisoned goats carrying fetuses of different ages. The RBC cholinesterase value of the nonpoisoned fetuses ran from 1.33 to 1.74. Compared with the above data, the blood of fetuses from lactating goats poisoned with parathion was depressed by about 85%. They investigated the milk production of three goats that received total doses ranging from 900 to 1,060 mg intramuscularly. The dosage for the goats on the milk test was 10 daily injections at a dose of 0.5 mg/kg followed by five daily injections at 1 mg/kg. These were then followed by five more daily injections at a dose of 2 mg/kg. They found no significant increase or decrease in milk production as compared with control periods that could be ascribed to the parathion treatment. The production of milk by all goats decreased sharply after initiating the final dosage, which was aimed at killing the animals in a short time.

In reference to reproductive studies, Beck (1953)[1/] investigated the effect of certain insecticides on the motility and metabolism of mammalian spermatozoa using boar semen. From oxygen-consumption experiments, the data indicated that DDT, aldrin, and malathion had a minor inhibitory effect on respiration of boar spermatozoa, whereas parathion, BHC, and TEPP had an intermediate effect. The insecticides had a pronounced effect on the motility of the spermatozoa. The motility was practically gone 120 min after parathion treatment. Furthermore, parathion had only a weak inhibitory effect on glycosis of spermatozoa.

Wild Avian Species - There is a large volume of literature on the effect of chlorinated hydrocarbons upon avian species. This interest has been

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[1/] Beck, S. D., "Effect of Insecticides on the Metabolism and Motility of Mammalian Spermatozoa," J. Econ. Entomol., 46:570-574 (1953).

largely due to the propensity for chlorinated hydrocarbons to accumulate in fatty tissue. The organic phosphorus insecticides degrade rapidly and have fewer latent effects than the more residual chlorinated hydrocarbons. Barnett (1950)[1] fed parathion to pheasants at levels of 100 and 1,000 ppm, and he noticed a decreased gonadal activity, and there was also a loss in total body weight. Shellenberger et al. (1966)[2] noted a similar weight loss in Japanese quail that were fed 5 ppm of Azodrin^{R}. Muller and Lockman (1972)[3] fed subtoxic doses of dieldrin and parathion to yearling mallards to observe the effect on egg production, fertility, hatchability, shell thickness, and progeny growth. Their test groups were fed 10 ppm of parathion in the diet. The treatment was started 30 days prior to egg production and continued for 90 days thereafter. Parathion failed to exert significant effects upon egg production, fertility, or progeny growth. Hatchability was depressed for those animals receiving dieldrin, but parathion had no effect. The only measurable effect of parathion was a significant reduction in mean shell thickness. The thinner shells do not restrict successful embryonic development. Neill et al. (1971)[4] conducted a similar study using the gray partridge as a test bird. These birds received 8 ppm of parathion. Here, again, parathion did not have an effect upon egg production, fertility, and hatchability. Eggs from the dieldrin treatment had a high percentage of dead-in-the-shell embryos which could be attributed to the residue of dieldrin that was found in the eggs. No parathion residues were detected in the eggs. The only effect noted from parathion in these studies was a high percentage of early and late dead embryos; the investigators hypothesized that undentified parathion degradation products may have been present in the eggs, resulting in an adverse effect on the metabolism, and thereby accounting for a high percentage of dead-in-the-shell embryos.

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[1] Barnett, D. C., "The Effect of Some Insecticide Sprays on Wildlife," *Proc. 30th Ann. Conf. West. Assoc. State Game and Fish Commissioners*, pp. 125-134 (1950).

[2] Shellenberger, T. E., G. W. Newell, R. F. Adams, and J. Barbaccia, "Cholinesterase Inhibition and Toxicologic Evaluation of Two Organophosphate Pesticides in Japanese Quail," *Toxicol. Appl. Pharmacol.*, 8:22-28 (1966).

[3] Muller, H. D., and D. C. Lockman, "Fecundity and Progeny Growth Following Subacute Insecticide Ingestion by the Mallard," *Poul. Sci.*, 51:239-241 (1972).

[4] Neill, D. D., H. D. Muller, and J. V. Shutze, "Pesticide Effects on the Fecundity of the Gray Partridge," *Bull. Environ. Contam. Toxicol.*, 6(6):546-551 (1971).

Furthermore, there was no alteration by either of the insecticides in the growth rate.

Teratogenic Effects

Mammals - It has been reported by Kimbrough and Gaines (1968)[1] that parathion can produce teratogenic effects in Sherman rats. The dosages were 3.0 and 3.5 mg/kg given intraperitoneally on the 11th day of pregnancy. The fetuses were removed on the 20th day of pregnancy. The results are shown in Table 14.

The average weight gain of the dams was drastically reduced; the number of fetuses was reduced by half. The number of resorptions was high and the weight of the fetuses from parathion-treated mothers was 30% less than the controls.

Fish (1966)[2] investigated the teratogenic potential of parathion and methyl parathion in rats. Dosage levels were chosen which represented about one-sixth, one-fourth, one-half, and three-fourths of the established oral or intraperitoneal LD_{50}. The doses were given intraperitoneally in a vehicle of ethanol (20%) and propylene glycol (80%). The injections were made on day 8, 9, 15, or 16 of gestation. In the search for anomalies, particular attention was directed to the external appearance of the embryo. Particular attention was given to the appearance of the brain, cervical and thoracic cord, heart and liver. Fish found no significant differences between control and treated animals for fetal mortality, fetal weight, or gross anomalies. Brain cholinesterase was reduced in the embryo brain. He did observe some large subcutaneous hematomas in the offspring of parathion-treated groups. The weight of the offspring from the parathion-treated animals was uniformly less than that of the offspring of the controls over the first 40 days of life. There was also a significant increase in stillbirths and neonatal deaths for the animals that were exposed to parathion. There has been a comment made in the Mrak Report (1969)[3] that Fish's (1966) data were like those observed with organochlorines. That is, the action of these two classes of insecticides suggest that they do not produce specific teratogenic effects.

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[1] Kimbrough, R. D., and T. B. Gaines, "Effect of Organic Phosphorus Compounds and Alkylating Agents on the Rat Fetus," Arch. Environ. Health, 16:805-808 (1968).

[2] Fish, S. A., "Organophosphorus Cholinesterase Inhibitors and Fetal Development," Am. J. Obstet. Gynecol., 96(8):1148-1154 (1966).

[3] Mrak, E. M., "Report on the Secretary's Commission on Pesticides and Their Relationship to Environmental Health," U.S. Dept. HEW, p. 665 (1969).

Table 14. THE TOXIC AND TERATOGENIC EFFECT OF PARATHION GIVEN INTRAPERITONEALLY TO RATS ON THE 11TH DAY OF PREGNANCY*

Compound	Dose (mg/kg)	Lowest dose to kill nonpregnant rat (mg/kg)	No.[a] pregnant rats	Average weight gain of dams (g)	Average no. fetuses per litter	Average no. dead fetuses per litter	Average no. resorptions per no. pregnant rats	Average[c] weight of fetuses (g)	Average weight of placentas (g)	No. and type of malformations per total no. fetuses
Control	--	--	4	67.78	10.8	0	0.5	3.57	0.69	None
Parathion	3.5	4	5	42.27	5.6	0.2	7.0	2.46[b]	0.53	1/28 fetus edematous
Control	--	--	5	75.24	12.6	0	0.8	3.34	0.54	None
Parathion	3.0	--	5	41.79[b]	7.6	0	3.8	2.86[b]	0.44[b]	None

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* Data from Kimbrough and Gaines, op. cit. (1968).

[a] In some pregnant rats all fetuses were resorbed, thus reducing the total numbers of litters.

[b] Significantly different from controls, $p < 0.05$.

[c] Weight of dead fetuses was included.

Existing data on the transfer of parathion through the placenta to the fetus are limited. Fischer and Plunger (1965)[1] did not find any traces of parathion in a human fetus from a mother who had taken a fatal dose of parathion. A study using pregnant guinea pigs also failed to demonstrate that parathion crosses the placenta. It has been shown by Fish (1966) that either methyl parathion or parathion given to pregnant rats inhibits brain cholinesterase activity in the embryos. Methyl paraoxon has been detected in the brain, liver, and muscle of the embryo (Ackermann and Engst, 1970[2]). Villeneuve et al. (1972)[3] used ^{14}C-parathion in a study of placental transfer in sheep. ^{14}C-parathion was administered to the dam over a 5- to 15-sec time interval using a jugular cannula. The dosage level was 0.1 mg/kg of body weight. Blood samples were drawn from the fetus, and the mother and amniotic fluid was collected at 5-min intervals initially and at longer periods of time thereafter for 48 hr. Plasma cholinesterase activity in the dam was maximally depressed within 30 min (52%). At 48 hr the plasma cholinesterase was still depressed by 30%. In the fetus, maximum depression occurred at 40 min and then plateaued at 20%, 4 hr after administration. After 48 hr the activity was depressed in the fetus 9.2%. Only a small portion (2 to 5%) of the radioactivity in the fetal plasma was in the form of parathion. The remainder of the radioactivity was in the form of unidentified metabolites. All the radioactivity from the parathion in the amniotic fluid was in the form of an unidentified metabolite. The results reported in this paper agree with other information on the metabolism of parathion in ruminant animals where the pathways are quite different from those of rodents or humans. It has been shown by O'Brien (1960)[4] that, in the cow, parathion is degraded to p-aminophenol and then conjugated to form a glucuronide. In humans and rodents, the major pathway involves the formation of paraoxon followed by hydrolysis to p-nitrophenol, or the direct hydrolysis of parathion to p-nitrophenol which is then glucuronidated and excreted in the urine.

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[1] Fischer, R., and C. Plunger, "Detection and Quantitative Determination of Phosphorus Insecticides in Biological Material," Mitt Arch. Toxicol., 21:101-105 (1965).

[2] Ackermann, H., and R. Engst, "Presence of Organophosphate Insecticides in the Fetus," Arch. Toxikol., 26:17-22 (1970).

[3] Villeneuve, D. C., R. F. Willes, J. B. Lacroix, and W. E. J. Phillips, "Placental Transfer of ^{14}C-Parathion Administered Intravenously to Sheep," Toxicol. Appl. Pharmacol., 21:542-548 (1972).

[4] O'Brien, R. D., Toxic Phosphorus Esters, New York: Academic Press, pp. 227-228 (1960).

Avian Embryotoxicity - There have been a number of reports of the toxicity of parathion on avian embryos (Khera and Bedok, 1967;[1] Khera and Lyon, 1968;[2] Khera and Clegg, 1969;[3] Roger et al., 1969;[4] Oga et al. 1971[5] and Yamada, 1972[6]). Khera and Bedok (1967) reported the following toxicity when 1 mg of parathion was injected into the egg sacs of 4-day incubated chicken eggs: the shape and course of the notochord was distorted. This distortion was associated with disproportionate and irregularly spaced anlagen of various axial structures. Khera and Lyon (1968) determined that chicken eggs injected on incubation days 0, 4, and 7, and duck eggs injected on days 0, 4, 7, and 10 were insuitable for toxicity studies. There was a large variation among replications, lack of dose-response relationships, and in some cases low sensitivity to the specific lethal effect. There was a more marked mortality response at mid-incubation for both species (10 days in chick and 13 days in duck embryos). There were indications that duck embryos provide less variable results than chick embryos. Roger et al. (1969) studied the teratogenic effect of bidrin in hen eggs. He included a number of other organophosphorus compounds in his study and reported on parathion. He observed that the injection of 1 mg of parathion/egg produced a definite shortening of the spine of the embryo. Thirty percent had abnormal feathers and 10% parrot-beak. When Oga et al. (1971) evaluated the teratogenic effects of parathion administered to eggs at levels 59.23, 118.46, or 296.15 µg/egg, he demonstrated dose dependency in the intensity of malformations. The highest dose-inoculated group showed a maximum incidence of teratogenic effects (44%) with treatment on the 8th day of embryogenesis. The embryos treated at the highest dose of parathion on the 8th day showed total body malformation, overall edema, ankylosis of the lower limbs, asymmetric implantation of the limbs, and intestinal atony. Embryos inoculated at 59.23 µg of parathion exhibited only limb malformations.

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[1] Khera, K. S., and S. Bedok, "Effects of Thiol Phosphates on Notochordal and Vertebral Morphogenesis in Chick and Duck Embryos," Food Cosmet. Toxicol., 5:359-365 (1967).

[2] Khera, K. S., and D. A. Lyon, "Chick and Duck Embryos in the Evaluation of Pesticide Toxicity," Toxicol. Appl. Pharmacol., 13:1-15 (1968).

[3] Khera, K. S., and D. J. Clegg, "Perinatal Toxicity of Pesticides," Can. Med. Assoc. J., 100:167-172 (1969).

[4] Roger, J-C., D. G. Upshall, and J. E. Casida, "Structure-Activity and Metabolism Studies on Organophosphate Teratogens and Their Alleviating Agents in Developing Hen Eggs with Special Emphasis on Bidrin," Biochem. Pharmacol., 18(2):373-392 (February 1969).

[5] Oga, S., C. C. Pellegatti, S. Reis, and A. C. Zanini, "Toxic and/or Teratogenic Activity of Anticholinesterase Compounds. I. Tetatogenicity of Parathion in Chicken Embryos," Rev. Farm. Bioquim. Univ. Sao Paulo, 9(2):343-355 (1971).

[6] Yamada, A., "Teratogenic Effects of Organophosphorus Insecticides in the Chick Embryo," Osaka Shiritsu Daigaku Igaku Zasshi, 21:245-255 (1972).

An effect on embryo development in hens' eggs was also reported by Marliac (1964).[1] A concentration of 0.1 mg of parathion per egg produced malformed embryos.

Lutz-Ostertag et al. (1969a, b),[2,3] Meiniel et al. (1970),[4] and Lutz-Ostertag et al. (1970)[5] studied the teratogenic effects of parathion in quail. Lutz-Ostertag et al. (1970) also investigated the development of pheasant, duck, and chick embryos as affected by parathion. The teratogenic effects of parathion on the skeleton of Japanese quail were studied by Meiniel et al. (1970). They immersed Japanese quail eggs in a mixture of acetone and parathion which contained 20 mg of parathion per liter of acetone. The immersion time was 30 sec. They observed inhibition of growth of the appendicular skeleton, which is considered a serious malformation as well as considerable retardation of the growth of the axial skeleton. The most common deformities were lordosis of the neck and numerous flexions in the sagittal plain. Neural centers and arches were evident in many of the cervical vertebrae. Also the vertebral musculature was disorganized. Nonincubated eggs of pheasant, quail, duck, and chickens were immersed in an acetone-parathion solution containing 20 g of parathion per liter (Lutz-Ostertag et al., 1970). Other eggs received injections of 1 g of commercial parathion (10% AI/liter) carried in 0.08 to 0.50 ml of Tyrodes solution. All of the embryos from eggs that were immersed and survived past 9 days of incubation were severely deformed. Deformation was exhibited by nanism, lordosis of the neck, celosomia, light edema, achondroplasia, anury, club-shaped featherbuds and abnormally positioned hind-legs with crisped digits were noticed. The livers were abnormal. The most interesting features were the changes in the urogenital tract. The gonads were partially or completely sterilized. Parathion had acted as an antimitotic substance, markedly affecting cell division. The Mueller's canals of the male embryos were partially retained. The right Mueller's canal in female embryos was completely retained. Quail embryonic testes were isolated from the embryo and cultivated (5 days) in vitro in the presence of pesticides (Lutz and Lutz-Ostertag, 1972).[1] The testes appeared healthy; however, upon histological examination there were changes in structure. The testicular tubules were vacuolized: the gonocytes and Sertoli cells disappeared completely. Quail embryo ovaries cultivated under the same conditions manifested similar changes. The effect on the chick embryo was not as broad. Only the selective disappearance of all the gonocytes was manifest. It further has been reported that the ovary of the chick embryo undergoes more or less complete suppression of the corta (Lutz-Ostertag and Meiniel, 1968).[2]

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[1] Marliac, J. P., "Toxicity and Teratogenic Effects of 12 Pesticides in the Chick Embryo," 48th Ann. Meeting Fed. Amer. Soc. Exp. Biol., 105 (1964).

[2] Lutz-Ostertag, Y., R. Meiniel, and H. Lutz, "Action du parathion sur le developpement de l'embryon de caille," C. R. Acad. Sci., Paris, Series D, 268:2911-2913 (1969a).

[3] Lutz-Ostertag, Y., R. Meiniel, and H. Lutz, "Effects du parathion sur le developpement de l'embryon de caille et de certains de ses organes *in vivo* et *in vitro*," Biol. Abstr., 52(22):12, 271 (1969b).

[4] Meiniel, R., Y. Lutz-Ostertag, and H. Lutz, "Teratogenic Effects of Parathion (Organophosphorus Insecticide) on the Skeleton of the Embryo of the Japanese Quail (Coturnix coturnix japonica)," Arch. Anat. Microsc. Morphol. Exp., 59(2):167-183 (1970).

[5] Lutz-Ostertag, Y., R. Meiniel, and H. Lutz, "Parathion, Embryonic Development, Sterilization and Estrogenic Effects in Birds; Comparison with the Effects of Aldrin," Anneé Biol., 9:501-507 (1970).

Dunachie and Fletcher (1969)[3] used the eggs of white leghorn chickens for teratogenic studies. They used the yolk injection method. Parathion was far less toxic when dissolved in acetone, compared to corn oil. Eighty-nine percent of the eggs hatched at the concentration of 50 ppm and only 19% at 500 ppm. Considering the high toxicity to mammals of parathion, the embryonic mortality was surprisingly low. The amount of deformities is variable but severe only at 500 ppm. Various combinations of parathion and malathion have been evaluated. Both mortality and deformities were high (38%) for parathion in a 5:50-ppm mixture with malathion. The hatchability of 100% parathion and no malathion was 65%, whereas a dose of 100% malathion reduced hatchability to 87%. A 50:50 mixture of these two organophosphate insecticides produced a hatchability of 71%.

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[1] Lutz, H., and Y. Lutz-Ostertag, "The Action of Different Pesticides on the Development of Bird Embryos," Drugs and Fetal Development, Proc. International Symposium on the Effect of Prolonged Drug Usage on Fetal Development, Beit-Berl, Kfar Saba, Israel, September 1971, pp. 127-150 (1972).

[2] Lutz-Ostertag, Y., and R. Meiniel, "In vitro Sterilizing Effect of Parathion on Embryonic Ovaries of Chick and Quail," C. R. Acad. Sci., Paris, Series D, 267:2178-2180 (1968).

[3] Dunachie, J. F., and W. W. Fletcher, "An Investigation of the Toxicity of Insecticides to Birds' Eggs Using the Egg-Injection Technique," Ann. Appl. Biol., 64(3):409-423 (1969).

Some very interesting observations have been made by Khera and Clegg (1969) relative to the prenatal toxicity of pesticides. These authors point out that the use of avian eggs as a test organism has a serious fault in that it is difficult to extrapolate to mammalian species. The greatest difference between the avian embryos and mammalian species is that the avian embryo is nonplacental. Since it is isolated from the maternal organism, the avian embryo cannot benefit from the natural defense mechanisms which safeguard the mammalian embryo. Furthermore, the phylogenetic relationship between avian species and man is remote. Mammalian data indicate that in most cases, extremely large dose levels (when compared to the human exposure levels) are required to induce neonatal effects or embryotoxicity. These investigators concluded it was unlikely that any pesticide presently in use is liable to cause damage to the perinatal human, at normal exposure levels. They felt that this position was supported by the lack of epidemiological data connecting pesticides to perinatal toxicity in humans.

Paraoxon was one of the compounds included in a study of Flockhart and Casida (1972)[1] in which they attempted to correlate the degree of inhibition of esterase in the yolk sac membrane to teratogenic effects of organophosphate insecticides.

The teratogenic effects were shown to be unrelated to hydrolysis of diphenylacetate by homogenates of yolk sac membranes. For example, both paraoxon and EPN gave similar degrees of esterase inhibition, but only paraoxon treatment of hens' eggs resulted in teratogenic effects.

Crustaceans - Pesticides may get into estuarine water by drainage from nearby treated fields, or when the pesticide is used as a control in salt marshes and estuaries for mosquitoes. Studies have been made by Butler et al. (1962)[2] on the effect of pesticides on adult oysters.

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[1] Flockhart, I. R., and J. E. Casida, "Relationship of the Acylation of Membrane Esterases and Proteins to the Teratogenic Action of Organophosphorus Insecticides and Eserine in Developing Hen Eggs," Biochem. Pharmacol., 21:2591-2603 (1972).

[2] Butler, P. A., A. J. Wilson, Jr., and A. J. Rick, "Effect of Pesticides on Oysters," Proc. Nat. Shellfish Assoc., 51:23-32 (1962).

Thirty-one compounds were investigated by Davis (1960)[1] as to their effect on fertilized eggs and larvae of bivalves. It was the opinion of these investigators that the effect of pesticides on growth is the most sensitive index for these molluscs for establishing a level that would (highest concentration) have no appreciable effect on the survival of embryos, or on the survival of the fully formed veliger larvae. Davis and Hidu (1969)[2] were interested in the effects of various compounds (52 compounds, including 17 insecticides) on the development of fertilized eggs of the hard clam, *Mercenaria mercenaria*, and the American oyster, *Crassostrea virginica*, and on the survival and growth of the larvae. Parathion concentrations in the water were 0.25, 0.50 and 1.0 ppm. They did not measure the number of oyster eggs developing or the survival of the larvae, but they did determine that the percent of increase in the length of the larvae was depreciated from 103% to 22% at the higher concentration. No evaluation was made for parathion for the reduction of the number of eggs ultimately developing into normal larvae from oyster and clam eggs.

Behavioral Effects

Al-Hachim and Fink (1968a, b, and c)[3-5] have studied the behavioral effects of DDT and parathion upon young mice whose mothers were previously treated with these compounds. They measured minimal electroshock seizure threshold (1968a) at 70 to 90 days of age, the condition of avoidance response (1968b) at 30 through 37 days of age, and open-field behavior (1968c) at 60 to 66 days. In all of these studies the mothers were given a dosage of 3 mg/kg of parathion during the first, second, and third trimester. There was no effect upon brain excitability, conditioned avoidance response, or open-field behavior of mice whose mothers had received the parathion. Out of the three tests, the only response that was obtained was from DDT which produced a delayed acquisition of controlled avoidance response. The brain excitability and the open-field behavior of the young were normal.

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[1] Davis, H. C., "Effects of Some Pesticides on Eggs and Larvae of Oysters (*Crassostrea virginica*) and Clams (*Venus mercenaria*)," *Commer. Fish. Rev.*, 23(12):8-23 (1960).

[2] Davis, H. C., and H. Hidu, "Effects of Pesticides on Embryonic Development of Clams and Oysters and on Survival and Growth of the Larvae," *Fish. Bull.*, 67(2):393-403 (1969).

[3] Al-Hachim, G. M., and G. B. Fink, "Effect of DDT or Parathion on the Minimal Electroshock Seizure Threshold of Offspring from DDT- or Parathion-Treated Mothers," *Psychopharmacol.*, Berlin, 13:408-412 (1968a).

[4] Al-Hachim, G. M., and G. B. Fink, "Effect of DDT or Parathion on Condition Avoidance Response of Offspring from DDT- or Parathion-Treated Mothers," *Psychopharmacol.*, Berlin, 12:424-427 (1968b).

[5] Al-Hachim, G. M., and G. B. Fink, "Effect of DDT or Parathion on Open Field Behavior of Offspring from DDT- or Parathion-Treated Mothers," *Psychol. Rept.*, 22:1193-1196 (1968c).

It was postulated that since parathion is metabolized and excreted by the mammals within a few days after ingestion, its effects on the central nervous system seem to be transient, having an effect for a few weeks after birth and disappearing when the offspring becomes older (2 months).

Spynu (1952 and 1957)[1,2] studied the conditioned reflexes of cats that had been dosed with 5, 3, and 2 mg/kg of parathion in three separate experiments (quoted by Medved et al., 1964[3]). At a dosage of 5 mg/kg orally, the conditioned reflexes of the cat were slowed within 10 to 15 min. The animal returned to normal in 3 days. After the 3 mg/kg dosage, it required 3 hr to establish a lowering of the conditioned relfexes. No changes could be found in animals given 2 mg/kg.

Toxicity Studies with Tissue Cultures

Tissue cultures were first used by Lewis and Richards (1945)[4] to determine the toxicity of DDT on chicken embryo tissues. Gabliks and Friedman (1965)[5] used HeLa cells and Chang liver cells in an attempt to

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[1] Spynu, E. I., "Data Concerning the Toxicology of the Insecticide Niuiph 100 and the Establishment of Tolerance Limits," Dissertation, Kiev (1952).

[2] Spynu, E. I., "The Effect of Some Organophosphorus Insecticides on the Higher Nervous Activities and on the Cholinesterase Activity," The Chemistry and Application of Organophosphorus Compounds, Acad. Sci. USSR (Ed.), Moscow (1957).

[3] Medved, L. I., E. I. Spynu, and Iu. S. Kagan, "The Method of Conditioned Reflexes in Toxicology and Its Application for Determining the Toxicity of Small Quantities of Pesticides," Residue Rev., 6:42-74 (1964).

[4] Lewis, W. H., and A. G. Richards, Jr., "Non-Toxicity of DDT on Cells in Cultures," Science, 102:330-331 (1945).

[5] Gabliks, J., and L. Friedman, "Responses of Cell Cultures to Insecticides. I. Acute Toxicity to Human Cells," Proc. Soc. Exp. Biol. Med., 120:163-168 (1965).

study insecticidal toxicity. The first evaluation of organophosphate insecticides in tissue cultures was conducted by Gabliks et al. (1967).[1] They found very little correlation between mouse liver cell cultures and human strain Chang liver cells. Litterst et al. (1969 and 1971)[2,3] used tissue cultures to investigate the toxicity of a number of insecticides including parathion, paraoxon, and p-nitrophenol. In the earlier study (1969) they also used HeLa cells. They evaluated growth inhibition and synthesis of proteins and nucleic acids in their cultures. They observed that organophosphate compounds were more toxic than the chlorinated compounds. It was found that paraoxon, an oxidation product of parathion, was less toxic at 40 ppm than parathion. In contrast, p-nitrophenol at the same concentration (40 ppm) appeared to be slightly more toxic. If they increased the treatment levels to 250 and 500 ppm, both paraoxon (at 250 ppm) and p-nitrophenol (at 500 ppm) caused nearly complete inhibition of cell growth. The synthesis of DNA at a concentration of 10 ppm of the insecticide indicated that only diazinon produced any changes. Aspirin and sodium chloride were included as positive controls in the test. The same effect observed with Diazinon® was produced by these two chemicals. It should be pointed out that a test level of 10 ppm is relatively high as related to concentrations of insecticides normally found in human tissues (with the exception of DDT). When the parathion concentration was raised to 50 ppm, there was a 50% inhibition in cell growth in 48 hr. Litterst and Lichtenstein (1971) extended the above studies to include the cells of nonmalignant origin. They chose normal diploid skin fibroblasts. They evaluated parathion, carbaryl, and several metabolites such as p,p'-DDE, paraoxon, p-nitrophenol, and 1-naphthol. The response to the normal cells was the same as previously reported with a malignant cell line, in that parathion was more toxic than paraoxon, and p-nitrophenol was as toxic as parathion. It should be emphasized that these results are exactly the opposite of data obtained when animals are used as test organisms. In general, little difference was found between the responses of the malignant (HeLa) and the nonmalignant (fibroblast) cells to the environmental toxicants. In review, it is difficult to make relevant predictions concerning the effect of a pesticide utilizing human cells in tissue culture. Under the conditions of the tests reported, the interaction at the level of synthesis of macromolecules is dependent upon the cell line and the additive used, and in general, there is no significant interaction between the two.

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[1] Gabliks, J., M. Bantug-Jurilla, and L. Friedman, "Responses of Cell Cultures to Insecticides. IV. Relative Toxicity of Several Organophosphates in Mouse Cell Cultures," Proc. Soc. Exp. Biol. Med., 125:1002-1005 (1967).

[2] Litterst, C. L., E. P. Lichtenstein, and K. Kajiwara, "Effects of Insecticides on Growth of HeLa Cells," J. Agr. Food Chem., 17: 1199-1203 (1969).

[3] Litterst, C. L., and E. P. Lichtenstein, "Effects and Interactions of Environmental Chemicals on Human Cells in Tissue Culture," Arch. Environ. Health, 22:454-459 (1971).

Mutagenic Effects

Dikshith (1973)[1] studied the *in vivo* effects of parathion on guinea pig chromosomes. He injected male guinea pigs with a single intratesticular injection of 0.05 mg of parathion in 0.5 ml of ethanol. The animals were killed after 24 hr. He determined the percentage of chromosomal changes at metaphase, and these results are shown in Table 15. Chromosome abnormalities were induced by parathion treatment in the meiotic chromosomes of the male guinea pig. In some cases, the individual morphology of the chromosomes was totally lost (chromosomal pulverization). Although it was not common, parathion did induce the formation of exchange figures. In essence, Dikshith's (1973) work confirmed the fact that cell division was inhibited at metaphase. It had been previously conjectured by Lutz-Ostertag et al. (1969a and 1969b) that parathion induces embryonic abnormalities because of defective cell division.

Oncogenic Effects

No ocogenic studies *per se* were found concerning parathion as an initiator of tumor development in animals. However, one study described the effect of parathion upon the possible reduction of tumors produced in animals by treatment with a potent carcinogen. No tumors were reported in the two year feeding studies.

Buchet and Lauwerys (1970 and 1971)[2,3] had observed that certain organophosphate esters inhibit tissue lipolysis *in vitro* and *in vivo*. Others have observed that mammary tumor incidence in rats appears to be influenced by the quality and the quantity of the dietary fat. Rats maintained on a diet containing 20% corn oil developed more mammary tumors after treatment with a single dose of 7,12-dimethylbenz(α)anthracene

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[1] Dikshith, T. S. S., "*In vivo* Effects of Parathion on Guinea Pig Chromosomes," *Environ. Physiol. Biochem.*, 3:161-168 (1973).

[2] Buchet, J. P., and R. Lauwerys, "Inhibition of Rat Heart Diolein Hydrolase and Brain Acetylcholinesterase by Organophosphate Esters *in vitro*," *Biochim. Biophys. Acta*, 218:369-371 (1970).

[3] Buchet, J. P., and R. Lauwerys, "Characterization of a Diglyceride Lipase in Rat Heart and Intestine," *Life Sci.*, 10:371-376 (1971).

Table 15. PERCENTAGE OF CHROMOSOMAL CHANGES AT METAPHASE OF MALE GUINEA PIGS AFTER PARATHION (0.05 MG/TESTICAL) TREATMENT*

Number of cells scored			Number of cells observed and percent frequency				
			Fragmentation		Clumping and liquification		
Control[a]	50% ethanol control	Para-thion	Cells	Percent	Cells	Percent	Other types
200	200	200	31	15.50	14	7.00	--
200	200[b]	200	28	14.00	19	9.50	--
200	200	200	23	11.50	15	7.50	--
200	200[b]	200	37	18.50	18	9.00	Observed Chromatid bridge in one cell
200	200	200	24	12.00	10	5.00	--
Average 200	200	200		14.30		7.60	--

Source: Dikshith, op. cit. (1973); reprinted from Environmental Physiology and Biochemistry by permission of the publisher. Year of first publication, 1973.

[a] Chromosomal changes were not noticed in any of the cells scored.

[b] One to two cells showed features of clumping otherwise all cells scored were normal.

than rats fed a low fat diet. Lauwerys and Buchet (1972)[1] evaluated the effect of parathion on mammary carcinoma produced by 7,12-dimethylbenz-(α)anthracene (DMBA). They injected parathion twice a week into rats for 12 weeks at a level of 0.5 mg/kg followed by 1 mg/kg intraperitoneally three times a week for 11 weeks. The triamiphos was included in the diet of another group of rats, at a concentration of 20 ppm. Two weeks after the beginning of the different treatments, all rats were given 0.5 ml of DMBA solution (5 mg) orally and the treatment was repeated for 5 weeks. Animals were autopsied at the time of death or at the end of the test period of 23 weeks. The carcinogenic action of DMBA observed during this study is summarized below.

Treatment group	Total tumor incidence	Number of tumors per tumor-bearing rat	Latent period in days
Parathion	100	2	66
Control	90	2.6	66
Triamiphos	60	1.6	73
Control	100	2.3	66

There was no reduction of tumor incidence in the parathion treated group. The lower tumor incidence in the triamiphos may be due to its reduction of tissue lipolytic activity. The administration of parathion may have allowed a return of normal lipolytic activity between successive injections.

Effects on Humans

Acute and Subacute Toxicity - There are extensive literature references to acute and subacute poisoning of humans by parathion, largely presented as individual or group clinical cases. In most of these instances the actual amounts of parathion ingested, absorbed or inhaled were not known. Hayes (1967)[2] has summarized the susceptibility of man and other animals to a number of pesticides, including parathion (Tables 16 and 17). Parathion is a highly toxic chemical with a fatal dose for man at a level of 2 mg/kg for a single dose. The oral lethal dose of parathion is

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[1] Lauwerys, R., and J. P. Buchet, "Effect of Two Organophosphorus Esters on Mammary Carcinogenesis by 7,12-Dimethylbenz(α)anthracene," Eur. J. Toxicol., 5:163-167 (1972).

[2] Hayes, W. J., Jr., "Toxicity of Pesticides to Man--Risks from Present Levels," Proc. R. Soc. Lond., 167(1007):101-127 (1967).

Table 16. SUSCEPTIBILITY OF MAN AND OTHER ANIMALS
TO SINGLE ORAL DOSES OF PARATHION

	Dosage (mg/kg)								
Species	Largest without Clinical Effect	Smallest with Clinical Effect	Median Clinical CD_{50}	Smallest with Serious Effect	Largest Nonfatal	Smallest Fatal	LD_{50}	Uniformly Fatal	References
Man	--	--	--	--	6.4	2·0	--	13	a/, b/
Child	--	--	--	--	--	0·1	--	--	c/
Calf	--	--	--	0·5	--	1·5	--	--	d/
Sheep	50	--	--	--	75	20	--	--	d/
Steer	25	--	--	--	--	--	--	--	d/

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a/ Goldblatt, M. W., "Organic Phosphorus Insecticides and the Antidotal Action of Atropine," Pharm. J., 164:229-233 (1950).

b/ Hayes, W. J., Jr., 1963 Clinical Handbook on Economic Poisons, PHS Publication No. 476, Washington, D.C. (1963).

c/ Kanagartatnam, K., W. H. Boon, and T. K. Koh, "Parathion Poisoning from Contaminated Barley," Lancet, 1:538-542 (1960).

d/ Radeleff et al., op. cit. (1955).

Table 17. THE SUSCEPTIBILITY OF MAN AND OTHER ANIMALS TO REPEATED ORAL DOSES OF PARATHION

Species	Dosage (mg/kg/day)	Duration (days)	Results	References
Man	0·1	42	33% reduction of whole blood ChE; 16% inhibition of RBC ChE; 37% inhibition of plasma ChE	a/
Rat (F)	0·26	84	80% reduction of RBC ChE; slight inhibition of plasma ChE	a/
Dog	0·047	168	60% inhibition of plasma ChE	b/
Pig	4·0	49	80% inhibition of RBC ChE; no inhibition of plasma ChE	a/

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a/ Edson et al., op. cit. (1964)
b/ Frawley and Fuyat, op. cit. (1957).

estimated to be 1.43 mg/kg of body weight for a man (DuBois, 1958).[1] As much as 0.1 mg/kg/day continuously administered for 42 days significantly reduced plasma and RBC cholinesterase. Lethality has been reported by Hayes (1963) following the ingestion by adult humans of 900, 120, and 50 mg of parathion.

Rider and his associates (1963, 1964, 1967, 1969a, b)[2-6] conducted a number of experiments on human volunteers from the San Quentin Prison in California to establish tolerances for various insecticides in man. Over a 10-year period, they worked with parathion, methyl parathion, malathion, EPN, Systox®, dichlorvos, OMPA, CARDONA, and Guthion®. Most all of their experiments were conducted over the same time frame, which was oral ingestion of a capsule of various dosages of the insecticides for a 30-day period. There was one exception (1963) in which the experiments ran for 50 days. Before the evaluations began, the prisoners were given physical examinations, and a base was established for their plasma and RBC cholinesterase. During the 30-day test, blood samples were taken twice a week for esterase activity. Rider and Moeller (1963) and Rider et al. (1969b) ran experiments using parathion on prisoners. Rider and Moeller (1963) and Rider et al. (1969a) reported on the effect of four dosage levels of parathion: 3.0, 4.5, 6.0 and 7.5 mg/day. They observed only a slight depression of plasma cholinesterase for levels of 3.0, 4.5 and 6.0 mg. However, at the level of 7.5 mg/day, on day 4 the plasma cholinesterase of one subject was down to 73% of his pretest level. On day 9 two of the subjects had values of 64% and 68% of their control levels, and on day 16 these two subjects had cholinesterase depressions of 50% and 52% of their pretest level. At this point in the experiment the average plasma cholinesterase for the five subjects was decreased by 28% from the control value. Some depression of red blood cell cholinesterase was also noted at the dosage of 7.5 mg of parathion per day. The lowest red cell cholinesterase values obtained were 63, 78, and 86% of the pretest levels. These data are in agreement with those of Edson et al. (1964).

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[1] DuBois, K. P., Postgrad. Med. J., 24:278-288 (1958).

[2] Rider, J. A., and H. C. Moeller, "Tolerance of Organic Phosphates in Man," Progress Report of Franklin Hospital Foundation, San Francisco, California (1 October 1963).

[3] Rider, J. A., and H. C. Moeller, "Studies on the Anticholinesterase Effects of Systox and Methyl Parathion in Humans," Fed. Proc., 23(2):176 (1964).

[4] Rider, J. A., H. C. Moeller, and E. J. Puletti, "Continuing Studies on Anticholinesterase Effect of Methyl Parathion, Initial Studies with Guthion, and Dichlorvos on Humans," Fed. Proc., 26(2):427 (1967).

[5] Rider, J. A., H. C. Moeller, E. J. Puletti, and J. I. Swader, "Toxicity of Parathion, Systox, Octamethyl Pyrophosphoramide, and Methyl Parathion in Man," Toxicol. Appl. Pharmacol., 14(3):603-611 (1969a).

[6] Rider, J. A., and E. J. Puletti, "Studies on the Anticholinesterase Effects of Gardona, Methyl Parathion, and Guthion in Human Subjects," Fed. Proc., 28(2):479 (1969b).

Symptoms of Parathion Poisoning - The symptoms of mild exposure to parathion as a result of orchard spraying or other activities associated in the fruit-growing industry have been described by Sumerford et al. (1953)[1] and Arterberry et al. (1961).[2] The modes of exposure and the symptomatology have been discussed by Hamblin and Golz (1955). The signs and symptoms of 246 patients admitted to a hospital in Greece with acute parathion poisoning have been reviewed by Tsachalinas et al. (1971).[3] Namba (1971)[4] has presented an excellent description of the signs and symptoms of organophosphate poisoning in patients. Reference should be made to Hamblin and Golz's paper (1955) for the onset and progressions of symptoms in subjects exposed to toxic amounts of parathion in spraying operations. Namba (1971) has classified the signs and symptoms observed in 77 patients who developed poisoning by the application of ethyl and methyl parathion. The more prominent symptoms were weakness, nausea or vomiting, excessive sweating, headache and excessive salivation. Namba points out that if the exposure to organic phosphorus insecticides is sufficient to produce symptoms, they usually appear in less than 12 hr. Symptomatology that appears 24 hr after exposure is unlikely to be due to these pesticides. A critical clinical observation is the occurrence of miosis, which is found in about 50% of the patients, and the latter symptom appears in subjects even in the mild cases. Death is usually attributed to failure of the respiratory muscles and paralysis of the respiratory center. Cardiac involvement may occur, but is usually seen only at the terminal stage. Man appears to be more sensitive to the organophosphate insecticides in that he exhibits symptoms earlier than experimental animals, particularly central nervous system manifestations. If an untreated organophosphate-poisoned victim is alive after 24 hr, he is likely to recover. The account by Kanagaratnam et al. (1960)[1] describes a parathion poisoning incident resulting from the use of contaminated barley in India. There were 53 persons involved, and the clinical features described included collapse, fits, sweating, dyspnoea, the effect on the pupils, the eye, blood pressure, coma, and muscular fasciculation.

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[1] Sumerford, W. T., W. J. Hayes, Jr., J. M. Johnston, K. Walker, and J. Spillane, "Cholinesterase Response and Symptomatology from Exposure to Organic Phosphorus Insecticides," AMA Arch. Ind. Hyg. Occup. Med., 7:383-398 (1953).

[2] Arterberry, J. D., W. F. Durham, J. W. Elliot, and H. R. Wolfe, "Exposure to Parathion," Arch. Environ. Health, 3:476-485 (1961).

[3] Tsachalinas, D., G. Logaras, and A. Paradelis, "Observations on 246 Cases of Acute Poisoning with Parathion in Greece," Eur. J. Toxicol., 4:46-49 (1971).

[4] Namba, T., "Cholinesterase Inhibition by Organophosphorus Compounds and Its Clinical Effects," Bulletin of the World Health Organization, 44:289-307 (1971).

Gershon and Shaw (1961)[2] felt that chronic exposure to organophosphate compounds produced psychiatric disorders in orchard workers. In a small field survey, they observed in 14 men and two women schizophrenic and depressive reactions with severe impairment of memory and difficulty in concentration. The range of exposure for these subjects was 1-1/2 to 10 years.

No other surveys of this nature were found in the literature.

Brown (1971)[3] reported on the electroencephalographic changes and disturbance of brain function following organophosphate exposure. Acute organophosphate poisoning disturbs central nervous system functions by causing disorientation in space and time, a sense of depersonalization, and hallucinations; with heavy exposure, convulsions occur. Acute inhibition of brain cholinesterase would be expected to cause effects related to the temporal lobe. EEG changes in acute organophosphate poisoning have been reported to resemble those seen in the interictal EEG of temporal lobe epileptics.

Dermal Effects - The information in this subsection is concerned with observations that were made during controlled exposure to parathion. Other data on the relationship of exposure to occupational hazards of dermal contact are discussed in a later subsection.

Fredriksson (1961a)[4] and Fredriksson et al. (1961) conducted a study on the distribution of labeled parathion within the skin, and on the hydrolysis and metabolism of this insecticide within the skin. Previous to their work, there had been no other studies using autoradiographic techniques to evaluate the cutaneous distribution of parathion which had been applied topically. Evaluations were made from skin samples taken from man, cat, rabbit and rat (Fredriksson, 1961a). Human skin was obtained at autopsy within 10 hr after death, and pieces of this skin were nailed to a board. Six microliters of ^{32}P-labeled parathion was then evenly distributed on an area of the skin of about 1.5 cm^2. The parathion was allowed to remain in contact with the skin for 1/2, 1, 2, 4 or 24 hr. After the exposure period, the parathion was usually blotted off the skin, and samples were frozen and examined by autoradiographic techniques. It was found that parathion appeared to penetrate to some extent into hair follicles and sebaceous glands, although no certain conclusions could be drawn regarding the routes of absorption. There was some increased activity below the epidermal layers. In another study (Fredriksson et al., 1961), the possible occurrence of skin enzymes capable of metabolizing parathion and paraoxon was investigated. Tissue slices of liver and skin were cut and the hydrolysis of parathion was measured by the Warburg method. It was found that parathion was not hydrolyzed or transformed into paraoxon by the skin of man, cats, rats or rabbits. Paraoxon was hydrolyzed by the skin of all species except rats, and the enzymatic action occurred at the fastest rate in the rabbit. The hydrolysis in rabbit skin amounted to about 20% of the paraoxon in 1 hr. In the skin from man and cat the rate of hydrolysis was about 1%.

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[1] Kanagartanam, K., W. H. Boon and T. K. Hoh, "Parathion Poisoning from Contaminated Barley, "Lancet 1:538-542 (1960).

[2] Gershon, S., and F. H. Shaw, "Psychiatric Sequelae of Chronic Exposure to Organophosphorus Insecticides," Lancet, pp. 1371-1374 (1961).

[3] Brown, H. W., "Electroencephalographic Changes and Disturbance of Brain Function Following Human Organophosphate Exposure," Northwest Med., 70:845-846 (1971).

[4] Fredriksson, T., "Studies on the Percutaneous Absorption of Parathion and Paraoxon. Part II. Distribution of ^{32}P-Labeled Parathion Within the Skin," Acta Derm. Venerol., 41:344-353 (1961a).

Hayes et al. (1964)[1] and Funckes et al. (1963)[2] investigated the excretion of p-nitrophenol and the depression of RBC cholinesterase in human volunteers that were subjected to exposure of parathion under strenuous circumstances.

Hayes et al. (1964) reported on three series of tests to evaluate the potential hazard of dermal exposure to parathion under controlled conditions.

Series 1 involved exposure of the hand and right forearm of volunteers to 2% parathion dust, 2% emulsion or 47.5% emulsifiable concentrates. After the toxicant was distributed over the hand and forearm, they were then secured in a polyethylene bag and placed in a constant temperature chamber. The treatments were as follows:

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[1] Hayes, G. R., Jr., A. J. Funckes, and W. V. Hartwell, "Dermal Exposure of Human Volunteers to Parathion," Arch. Environ. Health, 8:829-833 (1964).

[2] Funckes, A. J., G. R. Hayes, Jr., and W. V. Hartwell, "Urinary Excretion of Paranitrophenol by Volunteers Following Dermal Exposure to Parathion at Different Ambient Temperatures," J. Agr. Food Chem., 11:455-457 (1963).

1. Exposure to 5 g of 2% parathion dust for 2 hr on five successive days at 105°F.
2. Exposure to 4 liters of 2% parathion emulsion for 70 min at 81°F.
3. Exposure to 47.5% parathion emulsifiable concentrate for 120 min at 69°F.
4. Same as No. 3 except for 90 min at 100°F.

No significant changes in cholinesterase activity and no clinical signs were noted.

In Series 2, a volunteer exposed his entire body to 2% parathion dust. The dust was rubbed on the bare shoulders, back and chest, and then he put on a rubberized suit. Seven pounds of the dust was poured over the shoulders and down into the suit. The suit was closed and sealed. There were two exposures for 7.5 and 7 hr with an interval of 6 weeks between exposures. The temperature was 60°F. In a later test a male volunteer sat in a chamber, exposed totally except for his head, and was exposed to a vapor generated by heating 2% parathion powder to 200°F. The duration of exposure was 3 hr.

In the first test plasma cholinesterase activity was depressed to 44% of baseline at 24 hr after the initiation of the test, but was within 12% of normal within 120 hr. The volunteer in the second test did not experience symptoms of poisoning. The plasma cholinesterase was depressed to 82% of normal.

In Series 3, filter paper pads (900 cm^2) were allowed to absorb their capacity of equal values of parathion and ether. The pads were placed on the backs of volunteers and covered with plastic sheeting. Two volunteers were exposed twice for 3 hr to pads containing 40 to 50 g of parathion at temperatures of 54°, 79°, and 104°F.

Significant depression in cholinesterase activity of red blood cells did not occur. The cholinesterase activity was depressed 20% in plasma immediately after exposure at 104°F. There were no signs of poisoning.

The investigators commented that some individuals can sustain high dermal exposure to parathion without signs of poisoning or suffering severe depression of cholinesterase activity.

In another study (Funckes et al., 1963), data on the excretion of *p*-nitrophenol and blood cholinesterase activity were measured in volunteers exposed to parathion dust. The exposure consisted of weekly 2-hr applications of 2% parathion dust to two volunteers maintained at environmental temperatures ranging from 58° to 105°F. In these studies only the hand and forearm were exposed in a bag containing the test insecticide. These investigators found that there was a significant change in *p*-nitrophenol excretion in urine following dermal exposures at temperatures above 82°F.

More recently Maibach et al. (1971)[1] investigated the penetration of parathion, malathion, and carbaryl through the skin of volunteers by using labeled pesticides. The only variable in the experimental design was the anatomical site of application of the dose, which was regulated at $4\ \mu g/cm^2$ of skin. Applications of pesticide were made to the following anatomical regions: forearm, palm, foot (ball), abdomen, hand (dorsum), fossa cubitalis, scalp, jaw angle, postauricular, forehead, ear canal, axilla, and scrotum. They found that approximately 8.6% of parathion when applied to the forearm was absorbed, and all other absorption was referenced to the forearm data. Slightly more parathion penetrated the palm and the ball of the foot, whereas the abdomen and the dorsum of the hand permitted twice as much penetration as the forearm. The fossa cubitalis had twice the penetration of the forearm. The hairy areas such as the scalp, the angle of the jaw, the postauricular area and forehead had four times more penetration than the forearm, whereas the axilla had five times as much, and penetration from the scrotum was essentially complete.

Durham et al. (1972)[2] measured excretion of *p*-nitrophenol as an indicator for the degree of exposure of orchard spraymen to parathion. Both respiratory and dermal absorption were studied. From the evidence in their study they concluded that skin contamination is potentially more important as a route of absorption than the respiratory route although not necessarily more important in terms of poisoning. The data showed that much larger amounts of parathion impinge on the skin than are breathed in the lungs. These authors emphasize the importance (need) to protect the skin from exposure.

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[1] Maibach, H. I., R. J. Feldmann, T. H. Milby, and W. F. Serat, "Regional Variation in Percutaneous Penetration in Man," Arch. Environ. Health, 23:208-211 (1971).

[2] Durham, W. F., H. R. Wolfe, and J. W. Elliott, "Absorption and Excretion of Parathion by Spraymen," Arch. Environ. Health, 24:381-387 (1972).

It is understood that environmental factors will have an influence on the potential occupational hazards associated with use of insecticides. With the exception of studies with workers in pesticide plants, the bulk of information on exposure toxicity comes from studies carried out in outdoor work situations.

One study, however, was done in which greenhouse workers were observed during a 3-month exposure to parathion in a greenhouse. Additionally, determination of cholinesterase levels of these employees were made 5 weeks after the last application of insecticide (Culver et al., 1958[1/]). In their summary of findings these authors stated that on plant surfaces (in the greenhouse) parathion was present at initial concentration from 80 to 200 ppm and had an average half-concentration time of 80 hr. It was further shown that the red blood cell cholinesterase depression in the workers could not be definitely correlated to measured exposures to parathion although such a correlation was inferred. The red blood cell cholinesterase depression ranged from 15 to 20% and the size of this depression was not sufficient to elicit signs of intoxication.

Malkinson (1960)[2/] states that "Although the largest number of cases of toxic absorption (industrial poisoning) follows respiratory inhalation many industrial poisons are absorbed through the skin. . ."

Although poisoning with parathion often results from entry of the material by two or more routes (absorption through skin, respiratory tract, conjunctive or gastrointestinal tract) many instances have been reported where toxicity resulted primarily by absorption through the skin (Batchelor and Walker, 1954;[3/] Hamblin and Marchand, 1951[4/]).

[1/] Culver, D., J. Kinosian, W. Thielen, and R. Graul, "A Study of Exposure to Parathion in a Greenhouse," AMA Arch. Ind. Health, 18: 235-247 (1958).

[2/] Malkinson, F., "Percutaneous Absorption of Toxic Substances in Industry," AMA Arch. Ind. Health, 21:87-99 (1960).

[3/] Batchelor, G. S., and K. W. Walker, "Health Hazards Involving In-Use of Parathion in Fruit Orchards of North Central Washington," Arch. Ind. Hyg., 10:522 (1954).

[4/] Hamblin, D. O., and J. F. Marchand, "Parathion Poisoning," Am. Practitioner and Dig. Treatment, 2:1 (1951).

Inhalation Effects - Reports that assess the hazards from occupational exposure to parathion suggest that the majority of the occupational accidents are due to dermal exposure. The following information on inhalation is concerned with observations under controlled conditions. Other information on respiratory exposure is discussed under Occupational Exposure Hazards. Hartwell et al. (1964)[1] made a controlled study of respiratory exposure to volunteers from parathion. The volunteers were fitted with a breathing tube and a face mask which was attached to a toxic-vapor-generating chamber, in which parathion was placed in open trays as a liquid or powder and heated to various temperatures. The uptake of parathion through the breathing apparatus was not measured. In one series of tests on each of four consecutive days, exposure was maintained for 30 min to a vapor from 1 ml of fresh technical parathion spread over 36 in^2 and heated to 105° to 115°F. On the 5th day the amount of parathion was raised to 5 ml, spread over an 80 in^2 surface, and heated to 150°F. Under these conditions, after five exposures, the red blood cell cholinesterase was depressed by 98% and the activity of plasma cholinesterase was depressed by 83%. In another series, a volunteer breathed through a mask attached to the 6-ft x 6-ft x 8-ft plyboard chamber for 2-1/2 hr. In this chamber, panels of cheese-cloth 3 ft x 6 ft were saturated with technical parathion and attached to the interior walls. In addition, technical parathion was sprayed into the chamber. The temperature of the chamber was 100°F. The subject remained well after breathing air from this chamber. Based on p-nitrophenol excretion as an indicator, it was calculated that the average exposure amounted to 2.5 mg equivalents of parathion absorbed. After 2-1/2 hr the cholinesterase activity of the red blood cells was depressed about 24%. The investigators felt that their study confirmed the generally accepted respiratory exposure hazard afforded by parathion. Relating these levels to Edson's (1957)[2] work, where the latter demonstrated that cholinesterase activity was depressed 33% after ingested oral doses of 7.6 mg, Hartwell et al. (1964) felt that the hazard to respiratory exposure of humans to parathion was at least three times greater than ingestion.

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[1] Hartwell, W. V., G. R. Hayes, Jr., and A. J. Funckes, "Respiratory Exposure of Volunteers to Parathion," Arch. Environ. Health, 8: 820-825 (1964).

[2] Edson, E. F., "Effects of Prolonged Administration of Small Daily Doses of Parathion in Rat, Pig and Man," Mineograph Bulletin, Essex England: Chesterford Park Research Station NR Saffron Walden, p. 22 (March 1957).

Occupational and Accidental Exposure Hazards - The occupational hazards will be broadly considered as those exposures sustained by workers in field operations and workers in manufacturing operations.

Field operations - The occupational hazards of spraying parathion have been investigated by a number of workers. A very early paper by Williams and Griffiths (1951)[1] was concerned with the individual symptoms and cholinesterase level in the blood of sprayers. Others have reported specifically on the effects to humans in spraying operations only (Griffiths et al., 1951;[2] Ganelin et al., 1964;[3] Bick, 1967[4]). More complete studies involving all of the types of personnel that are connected with spraying operations such as field men, warehousemen, and residents in the area have been reported by Sumerford et al. (1953), Hayes et al. (1957),[5] Arterberry et al. (1961).

Milby and Ottoboni (1963)[6] reported on an epidemic of parathion poisoning is a 50 mile square area around Hughson, California. In this area there were 250 peach orchards with a total area of 24,000 acres. There were 5,000 to 8,500 peach pickers in this area between early August and mid-September. The peaches were picked manually in hot weather. During this season, 94 workers were poisoned by parathion. Cholinesterase determinations were obtained in 68 of the 94 cases and were found to be depressed in 66 patients. One death occurred which was attributed to parathion poisoning.

Two pounds of actual parathion were sprayed per acre (as a 25% wettable powder) on 20 March, 20 May, 25 June and 27 July. Picking was started on 8 August.

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[1] Williams, J. W., and J. T. Griffiths, "Parathion Poisoning in Florida Citrus Spray Operations," J. Fla. Med. Assoc., 37:707-709 (1951).

[2] Griffiths, J. T., C. R. Stearns, Jr., and W. L. Thompson, "Parathion Hazards Encountered Spraying Citrus in Florida," J. Econ. Entomol., 44:160-163 (1951).

[3] Ganelin, R. S., C. Cueto, and G. A. Mail, "Exposure to Parathion: Effect on General Population and Asthmatics," J. Amer. Med. Assoc., 188:807-810 (1964).

[4] Bick, M., "The Effect of Blood Cholinesterase Activity of Chronic Exposure to Pesticides," Med. J. Aust., 2:1066-1070 (27 May 1967).

[5] Hayes, W. J., Jr., E. M. Dixon, G. S. Batchelor, and W. M. Upholt, "Exposure to Organic Phosphorus Sprays and Occurrence of Selected Symptoms," Public Health Reports, U.S. Dept. HEW, Public Health Service, 72(9):787-794 (September 1957).

[6] Milby, T. H., and F. Ottoboni, "Report of an Epidemic of Organic Phosphate Poisoning in Peach Pickers, Stanisiaus County, California," State of California Department of Health, Bureau of Occup. Health (1963).

Analysis was performed for parathion residues on leaf and fruit samples. The contamination of workers' exposed skin was also determined. The air was sampled in three orchards in the breathing zone of the workers. These sources of information led to an estimate of the total dose by all routes of < 4,000 μg/day. The analyses performed by Milby and Ottoboni are summarized below.

Source	Parathion (μG)
Oral	
Four peaches per day (residue level of 0.5 ppm by weight)	500/day
Dermal	
Palms of hands (63 in^2 total and 7 $\mu g/in^2$ maximum)	440/day
Backs of hands, forearms and face ($351 in^2$ total and $4.7 \mu g/in^2$ total)	1,650/day
Neck ($40 in^2$ and $1.4 \mu g/in^2$ maximum)	56/day
Remainder of trunk	960/day
Respiratory	
Airborne dust ($35 \mu g/m^3$ and breathing rate of $10 m^3$/day)	350/day
Total dose	< 4,000/day

Data from Milby and Ottoboni, op. cit. (1963).

Quinby and Lemmon (1958)[1/] evaluated the effects of residues left in orchards on workers involved in picking, thinning, cultivating, and irrigating. Williams and Griffiths (1951), in a study of orchard sprayers, concluded that there should be a reassessment of the relationship of blood cholinesterase to the development of symptoms. It had been considered earlier than parathion poisoning would not be evident in such workers unless the blood cholinesterase level dropped to about 25% of normal. However, these workers observed symptoms of parathion poisoning when the plasma cholinesterase levels were as high as 90% of their control values, and the red blood cell cholinesterase was reduced to no

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[1/] Quinby, G. E., and A. B. Lemmon, "Parathion Residues as a Cause of Poisoning in Crop Workers," J. Amer. Med. Assoc., 166(7):740-746 (1958).

to no lower than 60 to 70% of the normal control values. In the five cases studied, four of the persons exhibited symptoms of poisoning when the red blood cell cholinesterase was between 50 and 75% of normal. Jegier (1964)[1] examined 52 subjects for dermal and respiratory exposure during a spraying season in the Provience of Quebec. It was found that the maximum exposure for parathion, malathion, Sevin®, endrin and Guthion® was less than 1% of the toxic dose. Spraying activities included air blast spraying of apple orchards and field spraying of vegetables and grain. A study of parathion poisoning cases reported during citrus spray operations in Florida orchards was reported in 1950 (Griffiths et al., 1951). Of the 48 cases reported, eight were shown to be definitely not parathion poisoning, and 15 others were highly questionable. The remaining 25 cases were assumed to be poison cases. However, a positive diagnosis was lacking because of the absence of blood tests for cholinesterase. These investigators suggested that skin absorption was the primary cause of parathion poisoning in these cases, and that handgun-spray operators appeared to have far more skin exposure than speed-sprayer operators. By checking the residues of parathion on the respirator filter discs, they obtained additional evidence in that the hand spray gun operators retained more parathion on their respiratory filter discs. An extensive study of symptomatology and cholinesterase response after exposure to organophosphorus insecticides has been reported by Sumerford et al. (1953). This study involved a total of 258 persons, 805 samples of red blood cells, and 802 samples of plasma which were analyzed for cholinesterase. The workers were divided into eight groups:

- Mixing plant personnel (MPP)
- Commercial applicators (CA)
- Part-time applicators (PTA)
- Workers in orchards (WO)
- Field men, warehousemen and miscellaneous workers (FWM)
- Residents living near orchards (RNO)
- Residents far from orchards (RFO)
- Residents outside area (ROA)

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[1] Jegier, Z., "Health Hazards in Insecticides Spraying of Crops," Arch. Environ. Health, 8:670-674 (1964).

This study was conducted in the apple growing area in Wenatchee, Washington, where two types of sprays were used, parathion and tetraethylpyrophosphate, and no effort was made to separate the effects of these two organic phosphate insecticides. This study was summarized with the following remarks:

1. The cholinesterase values for those groups known to have definite and consistent exposure (MPP, CA and PTA) were low, and the lowest cholinesterase values were exhibited by the mixing plant personnel (MPP).

2. As one would expect, those workers who had little or no exposure to insecticides maintained normal average cholinesterase values.

3. The only fatal or near fatal cases resulted from brief massive exposures and gross carelessness rather than long-standing exposure.

4. When an illness was characterized by miosis or by any three of a group of selected symptoms, it was possible to make a diagnosis of poisoning if, in addition, the cholinesterase values were significantly reduced.

They found no serious poisoning in the absence of excessive exposure or severe depletion of cholinesterase values. There were occurrences of mild illness in many of these workers when their cholinesterase values fell within normal ranges. A follow-up study was made in the same area by Hayes et al. (1957) during the subsequent 2-year period, with the subjects again grouped in the same order as described in the work reported above. Two hundred twenty-eight persons were involved in the follow-up study, and these included 100 of the same subjects studied previously. About the same number of persons were evaluated in 1953. In the latter study only commercial applicators, residents near orchards, residents far from orchards, and residents outside the area were compared. The results of the follow-up study confirmed the previous demonstrated relationships between blood cholinesterase levels, exposure and illness. It was again concluded that miosis or three or more selected symptoms are good criteria for the diagnosis of mild poisoning. No contradictions in results were found in the 1952 to 1953 test when compared to the 1951 observations. This paper also reported for the first time that symptoms resembling mild poisoning are not significantly more common in persons from an agricultural community than in persons living in a non-agricultural area. A third survey (Arterberry et al., 1961) was made in this same area in Wenatchee, Washington, and again involved several occupational groups that were studied in 1951, and in the 1952 to 1953 reports. About 115 persons were involved in the 1958 exposure study, 99 blood samples were analyzed for plasma and erythrocyte cholinesterase activity, and 571 urine samples were taken for p-nitrophenol content. The latter test was added because a sensitive procedure for p-nitrophenol determination had been developed and was later published by Elliott et al. (1960).[1] The results of the 1958 survey were summarized by stating that only the group with the highest potential exposure to parathion, that is the mixing plant personnel, sustained a definite decreased in cholinesterase activity during exposure. There was a significant excretion of p-nitrophenol during the period of exposure for mixing plant personnel, part-time ground applicators, commercial ground applicators, aircraft application workers and workers in orchards. Furthermore, the excretion of p-nitrophenol by residents near the orchard was not significant. They observed that p-nitrophenol excretion was elevated in all poisoning cases studied.

Bick (1967) in Australia made similar observations on the blood cholinesterase activity in orchard workers exposed to parathion and other organophosphorus insecticides and Sevin®. Bick collected blood samples at the beginning of the spray season in 1962 and 5 weeks later after a period of extensive spraying. In 1963 he again took samples before spraying and after spraying. He selected 15 orchard workers who had a marked decrease in plasma or red blood cell activity in 1962, and he tested these workers at intervals of 4 to 5 weeks for an additional 13 months, and a final collection was made 6 months later in September of 1965. He reported that the mean values of RBC cholinesterase activity for the orchard workers in 1962, before and after spraying, were significantly less than the value obtained on a control group who lives in a metropolitan area. After periods when no spraying activity was involved, the plasma and red blood cholinesterase activity increased. It was Bick's opinion that the use of the protective measures which had been recommended by responsible authorities reduced the exposure and was reflected by the minimal effect seen on red blood cell cholinesterase.

An interesting study was conducted by Ganelin et al. (1964) to determine if there were any deleterious effects on persons with respiratory disease that might be involved in exposure to organophosphorus compounds. This work was done in the Phoenix, Arizona, area, where it is known that a large number of people with respiratory and chronic diseases live. They used 122 subjects, grouped into five specific groups.

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[1] Elliott, J. W., K. C. Walker, A. E. Penick, and W. F. Durham, "A Sensitive Procedure for Urinary p-Nitrophenol Determination as a Measure of Exposure to Parathion," J. Agr. Food Chem., 8:111 (1960).

Group 1 - Nonasthmatics, no exposure to organic phosphate insecticides.

Group 2 - Nonasthmatics environmental exposure to organic phosphate insecticides.

Group 3 - Asthmatics, no exposure to organic phosphate insecticides.

Group 4 - Asthmatics, environmental exposure to organic phosphate insecticides.

Group 5 - Nonasthmatics, with occupational exposure to insecticides, which included the organic phosphate compounds.

The unexposed control subjects lived in Phoenix, and the environmental exposure groups all lived less than 500 yards from cotton fields treated with the insecticides. Ganelin et al. (1964) found no significant differences between asthmatics and nonasthmatics either in the exposed or unexposed groups. The p-nitrophenol excretion values were extremely low for all persons other than those in the occupational group.

Whereas there are a number of papers reported in the early 1950's on the occupational hazards of the use of organophosphorus insecticides in mixing and application by spraying, either from the ground or from the air, there are very few reports concerning the immediate effect of residual parathion in treated fields on workers or on establishment of adequate reentry intervals. Conley reported, in a scientific meeting in 1952, an episode where a number of workers in a vineyard which had been sprayed 33 days before with parathion became ill about 7 hr after work began (quoted by Quinby and Lemmon, 1958). Sixteen of the 24 developed symptoms requiring hospitalization. As far as is known, none had previous exposure to cholinesterase inhibitors. These workers suffered from weakness, diarrhea, miosis, headaches, and nausea. Symptoms were relieved with 1/100 g of atropine. There have been conflicting reports on the development of illness of workers in picking tobacco as related to insecticide residues. Lieben et al. (1953) recorded suspected parathion poisoning in tobacco pickers in Connecticut. Schaefer and Vance (1953)[1/] measured parathion residues on tobacco, and stated that

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[1/] Schaefer, R. A., and G. M. Vance, "Exposure of Connecticut Tobacco Workers to Parathion," AMA Arch. Ind. Hyg. Occup. Med.. 7:193-196 (1953).

few proved illnesses from skin absorption of parathion came to the attention of the Connecticut State Department of Health although there were thousands of workers handling tobacco leaves. Some interesting results have been obtained by Braid and Dustan (1955)[1/] on the persistence of parathion residues on immature peaches. They found that residues persisted on the fruit for 14 to 20 days and on the leaves for approximately 2 days. Bobb (1954)[2/] found that the half-life of parathion on peach tree bark was about 2 weeks, while only 2% of the residue persisted on the leaves after 1 week. Quinby and Lemmon (1958) investigated parathion poisoning in crop workers exposed to residues on pears, grapes, apples, hops, and citrus fruit in 11 different locations. Excellent detail is given in this paper on the symptoms and the types of exposure under many different situations. In some cases mild poisoning has been produced in workers thinning, picking, cultivating, and irrigating the aforementioned crops that were treated with 1 lb or more of parathion per acre. Several of the incidences of poisoning involved exposure to foliage or fruits sprayed not more than 2 days earlier. It is rather remarkable that contract with pear trees, citrus trees, and grapevines caused poisoning as much as 12, 17 and 33 days, respectively, after application of parathion. These investigators noted that in the group of outbreaks one common feature was that the foliage was chest-high. The main difference in the clinical picture between poisoning which resulted from contact with plants or from exposure during spraying and dusting was the relative mildness, the gradual onset, and the benign nature of the symptoms.

As has been reported by Ware et al (1973)[3/] the determination of safe reentry intervals is influenced by a number of factors including, (1) frequency and rate of application, (2) characteristics of the foliage, (3) height of the foliage, (4) density of the canopy, (5) the weather, and (6) inherent characteristics relative to the particular pesticide applied, i.e., persistance, toxicity, penetratability, etc. Other factors involved are length of exposure time, the type of clothing worn

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[1/] Braid, P. E., and G. G. Dustan, "Parathion Residues on Immature Peaches and the Hazard in Spraying and Thinning Operations," *J. Econ. Entomol.*, 48:44-46 (1955).

[2/] Bobb, M. L., "Parathion Residues on Peach Bark and Foliage," *J. Econ. Entomol.*, 47:190-191 (1954).

[3/] Ware, G. W., D. P. Morgan, B. J. Estesen, W. P. Cahill, and D. M. Whitacre, "Establishment of Reentry Intervals for Organophosphate-Treated Cotton Fields Based on Human Data: I. Ethyl and Methyl Parathion," *Arch. Environ. Contam. Toxicol.*, 1:48-50 (1973).

and presence or absence of respiratory protection. The recommended reentry time established must be long enough to give protection to the worker but short enough to be tenable with profitable agriculture practice.

In the study reported by Ware et al. (1973) on aerial application of a mixture containing 0.5 lb of methyl parathion, 0.5 lb of ethyl parathion and 2.0 lb of toxaphene in 5 gal. of spray per acre. The crop was mature cotton with bolls beginning to open. Ten minutes after application two men entered the field and collected samples for 30 min; this operation was repeated at 12, 24, 48, and 72 hr after insecticide treatment.

Biomedical data indicated that there was no clinical evidence of plasma or RBC cholinesterase depression or of urinary p-nitrophenol in either subject (man) even when the field was entered immediately after treatment.

Ware et al. (1973) reported that from the accumulation of residues on skin and clothing that an individual can expect exposure to the following amounts of mixed residues during a 30-min period at the treatment time indicated after treatment of a cotton field with methyl and ethyl parathion.

Time after treatment (hr)	Hands and forearms (mg)	Clothing (mg)	Inhalation (mg/ml)
0	3.47	18.21	1.06
12	1.93	12.11	0.60
24	1.16	6.57	0.36
48	0.60	4.52	0.18
72	0.31	2.65	0.09

These authors (Ware et al., 1973) suggest that with cotton, reentry can be safely made 12 to 24 hr after spraying with parathion.

Outstanding summaries of the exposure of workers to pesticides have been made by Wolfe et al. (1967)[1] and Hayes (1971).[2] Hayes (1971) has brought together (based on data from Wolfe et al., 1967), the potential exposure of workers to ethyl and methyl parathion, malathion, dichlorvos, chlorthion, carbaryl and azinphos-methyl. Information on parathion was shown (1) for air-blast spraying fruit orchards, (2)air-blast spraying orchards with concentrate, (3) spraying orchards with high-pressure powered handguns, and (4) spraying tomato plants with mist from hand-operated knapsack sprayer. The highest exposure (respiratory) to parathion was for spraying orchards with high-pressure powered handguns. The greatest exposure (dermal) to parathion was air-blast spraying of fruit orchards.

Hayes (1971) expressed the thinking that, although the concentrations of pesticides in working areas very often exceed threshold limit values, the reported concentrations are not necessarily dangerous. The threshold limit values for industrial chemicals of comparative toxicity are usually higher than that for pesticides. A statement was made by Hayes that about 80% of the total absorption of parathion is by the dermal route under orchard spraying conditions. Hayes (1971) summarized factors influencing respiratory and dermal exposure:

1. Type of formulation.
2. The concentration of the formulation.
3. The method of application.
4. The duration of application.
5. The type of work.
6. The wind and other environmental factors.
7. The attitude of the workers.

As one would expect, the variations of the exposure are wide (Wolfe et al, 1967); dermal exposure could range as much as 200-fold and the respiratory exposure in the form of concentrate might range up to 300-fold. The type of spraying is also influential. Spraying into trees affords much more exposure to the operator than similar spraying on row crops. Wind speed is so important that some states have laws specifying the highest velocity for application of certain types of herbicides. Another environmental factor is temperature. It has been shown (Funckes et al., 1963) that temperature affects the rate of absorption through the skin.

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[1] Wolfe, H. R., W. F. Durham, and J. F. Armstrong, "Exposure of Workers to pesticides," Arch. Environ. Health, 14:622-633 (1967).

[2] Hayes, W. J., Jr., "Studies on Exposure During the Use of Anticholinesterase pesticides, "Bulletin of the World Health Organization, 44:277-288 (1971).

The potential for oral exposure from eating food items contaminated with parathion as a result of handling with unwashed hands was evaluated in a study by Armstrong et al. (1973).[1] Two situations were considered: (1) the effect of drift during spray operations on food items carried by workers and (2) the contamination of foods by the hands of workers who had been exposed to treated foliage, etc. (fruit thinners).

The results of this study indicated that the potential exposure resulting from contamination of food items by unwashed hands is not high under normal conditions of apple thinning or in the application of dilute spray solutions. The daily intake of parathion under the conditions described above was reported to be below 3 mg/day.

Higher intakes of insecticides by hand contamination of snack items can be expected where sprayers carry food items such as candy bars and where concentrated formulations are being handled.

Wolfe et al. (1967) indicated that the morbidity of persons who are exposed from occupational standpoint is not great because (1) the dangerous operations only occupy a few hours a day or a week; (2) the workers usually wear protective clothing; and (3) the absorption by workers in the field is not comparable to exposures administered to laboratory animals.

Manufacturing operations - The number of studies conducted on the relationship of the manufacturing and packaging of parathion as to industrial hazards is limited.

Brown and Bush (1950)[2] made a study of parathion in the atmosphere of an industrial plant which manufactured the concentrated material.

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[1] Armstrong, J. F., H. R. Wolfe, S. W. Comer, and D. C. Staiff, "Oral Exposure of Workers to Parathion Through Contamination of Food Items," Bull. Environ. Contam. Toxicol., 10:3218-3327 (December 1973).

[2] Brown, H. V., and A. F. Bush, "Parathion Inhibition of Cholinesterase," Arch. Ind. Hyg. Occup. Med., 1:633-636 (1950).

They placed air samplers at the sites of different operations to measure the parathion. In addition, the plasma and red blood cell cholinesterase of the persons in the plant were determined. The evaluations for the red blood cell cholinesterase were made over a period of 1 year at intervals of 2, 3, and 5 months. The subjects were chemists, janitors, utility men, engineers, supervisors, workers in mixing rooms, handymen, and the plant superintendent. The data indicated that they felt that a physiological effect was produced. Although the air samples showed a maximum concentration of only 8 mg of parathion per 10 m^3 of air, they felt that these concentrations may be potentially dangerous if breathed over a long period of time. Another in-plant study was made in later years by Hartwell and Hayes (1965).[1/] Using two plant sites as a test base, these investigators compared the effectiveness of certain respiratory protection. Plant A's ventilation system, was found to be deficient. Plant B was a modern facility. The general ventilation was considered quite adequate, and only nontoxic dusts were handled manually. Each formulating plant processed phosdrin, methyl parathion, and ethyl parathion. They both evaluated poisoning by clinical signs as well as a depression of the cholinesterase activity of 20% or more. During the first year Plant A sustained 17 cases of alleged poisoning, and depression in cholinesterase activity of 20% was observed 41 times among 26 subjects. This load of toxicity led to the installation of a system to distribute uncontaminated compressed air to workers engaged in hazardous operations. After these alterations no cases of poisoning occured, and depressions in cholinesterase activity were observed only four times among 13 workers. Plant B, which theoretically afforded better protection, had to shut down for 10 days because of the occurence of poisoning. The fault was due to a compresser that both supplied air to the operators and forced concentrates into the mixing chamber. When a separate compressor was used for each operation, no cases of poisoning were observed.

Accidents - Parathion is one of the pesticides most frequently cited in incidents involving accidental exposure to pesticides. Preliminary data from the EPA Pesticide Accident Surveillance System (PASS) shows that parathion is the third most frequently cited pesticide in 1973. Based on an analysis of PASS data, Osmun (1974)[2/] stated that for 1972 and 1973, parathion and/or methylparathion were connected with 78% of the reported episodes relating to agricultural jobs, particularly those involving fields sprayed with pesticides for which safe reentry times for workers had been set.

[1/] Hartwell, W. V., and F. R. Hayes, Jr., "Respiratory Exposure to Organic Phosphorus Insecticides," Arch. Environ. Health, 11:564-568 (1965).

[2/] Osmun. J. V., "PASS Information Relating to Agriculture Jobs," Internal EPA Memo to Ed Johnson (1 April 1974).

There are a number of limitations in attempting to use PASS data. First of all, the cause-effect relationship between the pesticide(s) cited and the effects observed have generally not been established. Secondly, only data for 1972 through about January 1974 has generally been computerized and readily available for retrieval. Thirdly, a large portion of the data provided to PASS comes from California (e.g., 95 of the 257 parathion episodes involving humans, animals, plants, and area contamination in the computerized data base). This skewed distribution is probably a result of the method used by California to document pesticide information. In addition, files on the investigations made to follow up on episodes reported in California are not part of the PASS data base. Information in addition to that found on the pesticide episode reporting form was available on only 12 of the 257 episodes involving parathion.

On 10 June 1974, protection standards for agricultural workers in fields treated with parathion became effective (Quarles, 1974)1/. These standards prohibit application of parathion when unprotected workers are in the area being treated and require that unprotected workers not enter fields treated with pesticides for at least 48 hr.

1/ Quarles, J., "Worker Protection Standards for Agricultural Pesticides," Federal Register, 39(92):16888-16891 (10 May 1974).

References

Ackermann, H., and R. Engst, "Presence of Organophosphate Insecticides in the Fetus," Arch. Toxikol., 26:17-22 (1970).

Ahmed, M. K., J. E. Casida, and R. E. Nichols, "Bovine Metabolism of Organophosphorus Insecticides: Significance of Rumen Fluid with Particular Reference to Parathion," J. Agr. Food Chem., 6:740-746 (1958).

Alary, J-G., and J. Brodeur, "Correlation Between the Activity of Liver Enzymes and the LD_{50} of Parathion in the Rat," Can. J. Physiol. Pharmacol., 48:829-831 (1970).

Aldridge, W. N., "Serum Esterases, 2. An Enzyme Hydrolysing Diethyl p-Nitrophenyl Phosphate (E600) and Its Identity with the A-Esterase of Mammalian Sera," Biochem. J., 53:117-124 (1953).

Al-Hachim, G. M., and G. B. Fink, "Effect of DDT or Parathion on the Minimal Electroshock Seizure Threshold of Offspring from DDT or Parathion-Treated Mothers," Psychopharmacol., Berlin, 13:408-412 (1968a).

Al-Hachim, G. M., and G. B. Fink, "Effect of DDT or Parathion on Condition Avoidance Response of Offspring from DDT or Parathion-Treated Mothers," Psychopharmacol., Berlin, 12:424-427 (1968b).

Al-Hachim, G. M., and G. B. Fink, "Effect of DDT or Parathion on Open Field Behavior of Offspring from DDT- or Parathion-Treated Mothers," Psychol. Rept., 22:1193-1196 (1968c).

Andersen, A. A., and O. Karlog, "Elimination of Parathion in Cows After Oral and Dermal Administration," Acta Vet. Scand., 4:156-169 (1963).

Anon., "Toxic Hazards of Pesticides to Man. Report of a Study Group," WHO Tech. Rept. Ser., 114:3-51 (1956).

Anon., Council of Europe-Netherlands Report on Parathion and Parathion-Methyl, Ministerie von Sociale Zahen En Volksgenzandheid (1964).

Armstrong, J. F., H. R. Wolfe, S. W. Comer, and D. C. Staiff, "Oral Exposure of Workers to Parathion Through Contamination of Food Items," Bull. Environ. Contam. Toxicol., 10:321-327 (December 1973).

Arterberry, J. D., W. F. Durham, J. W. Elliot, and H. R. Wolfe, "Exposure to Parathion," Arch. Environ. Health, 3:476-485 (1961).

Baeza, C., A. M. Goldberg, and R. J. Rubin, "Effect of Carbon Monoxide on Response to Parathion and Paraoxon," Toxicol. Appl. Pharmacol., 22(2):288 (1972).

Barnes, J. M., and F. A. Denz, "The Chronic Toxicity of p-Nitrophenyl Diethyl Thiophosphate (E. 605): A Long-Term Feeding Experiment with Rats," J. Hyg., 49:430-441 (1951).

Barnett, D. C., "The Effect of Some Insecticide Sprays on Wildlife," Proc. 30th Ann. Conf. West. Assoc. State Game and Fish Commissioners, pp. 125-134 (1950).

Bass, S. W., A. J. Triolo, and J. M. Coon, "Effect of DDT on the Toxicity and Metabolism of Parathion in Mice," Toxicol. Appl. Pharmacol., 22:684-693 (1972).

Batchelor, G. S., and K. W. Walker, "Health Hazards Involving In-Use of Parathion in Fruit Orchards of North Central Washington," Arch. Ind. Hyg., 10:522 (1954).

Beck, S. D., "Effect of Insecticides on the Metabolism and Motility of Mammalian Spermatozoa," J. Econ. Entomol., 46:570-574 (1953).

Benke, G. M., K. L. Cheever, F. E. Mirer, and S. D. Murphy, "Comparative Toxicity, Anticholinesterase Action and Metabolism of Methyl Parathion in Sunfish and Mice," Toxicol. Appl. Pharmacol., 28:97-109 (1974).

Bick, M., "The Effect of Blood Cholinesterase Activity of Chronic Exposure to Pesticides," Med. J. Aust., 2:1066-1070 (27 May 1967).

Bobb, M. L., "Parathion Residues on Peach Bark and Foliage," J. Econ. Entomol., 47:190-191 (1954).

Braid, P. E., and G. G. Dustan, "Parathion Residues on Immature Peaches and the Hazard in Spraying and Thinning Operations," J. Econ. Entomol., 48:44-46 (1955).

Brodeur, J., and K. P. DuBois, "Comparison of Acute Toxicity of Anticholinesterase Insecticides to Weanling and Adult Male Rats," Proc. Soc. Exp. Biol. Med., 114(2):509-511 (November 1963).

Brown, H. V., and A. F. Bush, "Parathion Inhibition of Cholinesterase," Arch. Ind. Hyg. Occup. Med., 1:633-636 (1950).

Brown, H. W., "Electroencephalographic Changes and Disturbance of Brain Function Following Human Organophosphate Exposure," Northwest Med., 70:845-846 (1971).

Buchet, J. P., and R. Lauwerys, "Inhibition of Rat Heart Diolein Hydrolase and Brain Acetylcholinesterase by Organophosphate Esters *in vitro*," Biochim. Biophys. Acta, 218:369-371 (1970).

Buchet, J. P., and R. Lauwerys, "Characterization of a Diglyceride Lipase in Rat Heart and Intestine," Life Sci., 10:371-376 (1971).

Butler, P. A., A. J. Wilson, Jr., and A. J. Rick, "Effect of Pesticides on Oysters," Proc. Nat. Shellfish Assoc., 51:23-32 (1962).

Cook, J. W., "*In vitro* Destruction of Home Organophosphates Pesticides by Bovine Rumen Fluid," J. Agr. Food Chem., 5(11):859-863 (1957).

Culver, D., J. Kinosian, W. Thielen, and R. Graul, "A Study of Exposure to Parathion in a Greenhouse," AMA Arch. Ind. Health, 18:235-247 (1958).

Dahm, P. A., F. C. Fountaine, J. C. Pankaskie, R. C. Smith, and F. W. Atkeson, "The Effects of Feeding Parathion to Dairy Cows," J. Dairy Sci., 33(101):747-757 (1950).

Dahm, P. A., "Some Aspects of the Metabolism of Parathion and Diazinon," in Biochemical Toxicology of Insecticides, P. D. O'Brien and I. Yamamoto (eds.), New York: Academic Press (1970).

Dalloz, J. P., Delatour, and G. Lorgue, "The Organophosphorus Pesticides," Rev. Med. Vet., Toulouse, 123(10):1356 (1972).

Davis, H. C., "Effects of Some Pesticides on Eggs and Larvae of Oysters (*Crassostrea virginica*) and Clams (*Venus mercenaria*)," Commer. Fish. Rev., 23(12):8-23 (1960).

Davis, H. C., and H. Hidu, "Effects of Pesticides on Embryonic Development of Clams and Oysters and on Survival and Growth of the Larvae," Fishery Bull., 67(2):393-403 (1969).

Davison, A. N., "The Conversion of Schradan (OMPA) and Parathion into Inhibitors of Cholinesterase by Mammalian Liver," Biochem. J., 61:203-209 (1955).

Deichmann, W. B., W. Pugliese, and J. Cassidy, "Effects of Dimethyl and Diethyl Paranitrophenyl Thiophosphate on Experimental Animals," AMA Arch. Ind. Hyg. Occup. Med., 5:44-51 (1952).

Dikshith, T. S. S., "In vivo Effects of Parathion on Guinea Pig Chromosomes," Environ. Physiol. Biochem., 3:161-168 (1973).

DuBois, K. P., J. Doull, P. R. Salerno, and J. M. Coon, "Studies on the Toxicity and Mechanism of Action of p-Nitrophenyl Diethyl Thionophosphate (Parathion)," J. Pharmacol. Exp. Ther., 95(1):79-91 (1949).

DuBois, K. P., Postgrad. Med. J., 24:278-288 (1958).

DuBois, K. P., and F. K. Kinoshita, "Influence of Induction of Hepatic Microsomal Enzymes by Phenobarbital on Toxicity of Organic Phosphate Insecticides," Proc. Soc. Exp. Biol. Med., 129:699-702 (1968).

DuBois, K. P., "The Toxicity of Organophosphorus Compounds to Mammals," Bulletin of the World Health Organization, 44:233-240 (1971).

Dunachie, J. F., and W. W. Fletcher, "An Investigation of the Toxicity of Insecticides to Birds' Eggs Using the Egg-Injection Technique," Ann. Appl. Biol., 64(3):409-423 (1969).

Durham, W. F., H. R. Wolfe, and J. W. Elliott, "Absorption and Excretion of Parathion by Spraymen," Arch. Environ. Health, 24:381-387 (1972).

Ecobichon, D. J., and W. Kalow, "Action of Organophosphorus Compounds Upon Esterases of Human Liver," Can. J. Biochem. Physiol., 41:1537-1546 (1963).

Edson, E. F., "Effects of Prolonged Administration of Small Daily Doses of Parathion in Rat, Pig and Man," Mimeograph Bulletin, Essex England; Chesterford Park Research Station NR Saffron Walden, p. 22 (March 1957).

Edson, E. F., and D. N. Noakes, "The Comparative Toxicity of Six Organophosphorus Insecticides in the Rat," Toxicol. Appl. Pharmacol., 2:523-539 (1960).

Edson, E. F., et al., "Summaries of Toxicological Data: No-Effect Levels of Three Organophosphates in the Rat, Pig, and Man," Food Cosmet. Toxicol., 2:311 (1964).

Edson, E. F., D. M. Sanderson, and D. N. Noakes, "Acute Toxicity Data for Pesticides (1964)," World Rev. Pest Con., 4:36-41 (1965).

Elliott, J. W., K. C. Walker, A. E. Penick, and W. F. Durham, "A Sensitive Procedure for Urinary p-Nitrophenol Determination as a Measure of Exposure to Parathion," J. Agr. Food Chem., 8:111 (1960).

Fischer, R., and C. Plunger, "Detection and Quantitative Determination of Phosphorus Insecticides in Biological Material," Mitt Arch. Toxicol., 21:101-105 (1965).

Fish, S. A., "Organophosphorus Cholinesterase Inhibitors and Fetal Development," Am. J. Obstet. Gynecol., 96(8):1148-1154 (1966).

Flockhart, I. R., and J. E. Casida, "Relationship of the Acylation of Membrane Esterases and Proteins to the Teratogenic Action of Organophosphorus Insecticides and Eserine in Developing Hen Eggs," Biochem. Pharmacol., 21:2591-2603 (1972).

Frawley, J. P., E. C. Hagan, and O. G. Fitzhugh, "A Comparative Pharmacological and Toxicological Study of Organic Phosphate-Anticholinesterase Compounds," J. Pharmacol. Exp. Ther., 105:156-165 (1952).

Frawley, J. P., and H. N. Fuyat, "Effect of Low Dietary Levels of Parathion and Systox on Blood Cholinesterase of Dogs," J. Agr. Food Chem., 5:346 (1957).

Fredriksson, T., W. L. Farrior, Jr., and R. F. Witter, "Studies on the Percutaneous Absorption of Parathion and Paraoxon. Part I. Hydrolysis and Metabolism Within the Skin," Acta Derm. Venerol., 41:335-343 (1961).

Fredriksson, T., "Studies on the Percutaneous Absorption of Parathion and Paraoxon. Part II. Distribution of ^{32}P-Labelled Parathion Within the Skin," Acta Derm. Venerol., 41:344-352 (1961a).

Fredriksson, T., "Studies on the Percutaneous Absorption of Parathion and Paraoxon. Part III. Rate of Absorption of Parathion," Acta Derm. Venerol., 41:353-363 (1961b).

Fredriksson, T., "Studies on the Percutaneous Absorption of Parathion and Paraoxon. Part VI. In vivo Decomposition of Paraoxon During the Epidermal Passage," J. Invest. Dermatol., 42:37-40 (1964).

Fukuto, T. R., and R. L. Metcalf, "Metabolism of Insecticides in Plants and Animals," Ann. N. Y. Acad. Sci., 160:97-113 (1969).

Funckes, A. J., G. R. Hayes, Jr., and W. V. Hartwell, "Urinary Excretion of Paranitrophenyl by Volunteers Following Dermal Exposure to Parathion at Different Ambient Temperatures," J. Agr. Food Chem., 11:455-457 (1963).

Gabliks, J., and L. Friedman, "Responses of Cell Cultures to Insecticides. I. Acute Toxicity to Human Cells," Proc. Soc. Exp. Biol. Med., 120: 163-168 (1965).

Gabliks, J., M. Bantug-Jurilla, and L. Friedman, "Responses of Cell Cultures to Insecticides. IV. Relative Toxicity of Several Organophosphates in Mouse Cell Cultures," Proc. Soc. Exp. Biol. Med., 125: 1002-1005 (1967).

Gage, J. C., "A Cholinesterase Inhibitor Derived from O,O-Diethyl O-p-nitrophenyl Thiophosphate *in vivo*," Biochem. J., 54:426-430 (1953).

Gagne, J., and J. Brodeur, "Metabolic Studies on the Mechanisms of Increased Susceptibility of Weanling Rats to Parathion," Can. J. Physiol. Pharmacol., 50:902-915 (1972).

Gaines, T. B., "The Acute Toxicity of Pesticides to Rats," Toxicol. Appl. Pharmacol., 2:88-99 (1960).

Gaines, T. B., W. J. Hayes, Jr., and R. E. Linder, "Liver Metabolism of Anticholinesterase Compounds in Live Rats: Relation to Toxicity," Nature, 209(5018):88-89 (1966).

Gaines, T. B., "Acute Toxicity of Pesticides," Toxicol. Appl. Pharmacol., 14:515-534 (1969).

Ganelin, R. S., C. Cueto, and G. A. Mail, "Exposure to Parathion: Effect on General Population and Asthmatics," J. Amer. Med. Assoc., 188:807-810 (1964).

Gershon, S., and F. H. Shaw, "Psychiatric Sequelae of Chronic Exposure to Organophosphorus Insecticides," Lancet, pp. 1371-1374 (1961).

Giachetti, A., C. Grasso, and G. Bernardi, "Persistence of O,O-Diethyl-O-para-nitrophenyl phosphate (Paraoxon) in the Brain of the White Rat Treated with a Single, Subtoxic Dose of Parathion," Ric. Sci., 36(10): 1077 (1966).

Goldblatt, M. W., "Organic Phosphorus Insecticides and the Antidotal Action of Atropine," Pharm. J., 164:229-233 (1950).

Griffiths, J. T., C. R. Stearns, Jr., and W. L. Thompson, "Parathion Hazards Encountered Spraying Citrus in Florida," J. Econ. Entomol., 44:160-163 (1951).

Hamblin, D. O., and H. H. Golz, "Parathion Poisoning, A Brief Review," Ind. Med. Surg., 24(22):65-72 (1955).

Hamblin, D. O., and J. F. Marchand, "Parathion Poisoning," Am. Practitioner and Dig. Treatment, 2:1 (1951).

Hartwell, W. V., G. R. Hayes, Jr., and A. J. Funckes, "Respiratory Exposure of Volunteers to Parathion," Arch. Environ. Health, 8:820-825 (1964).

Hartwell, W. V., and G. R. Hayes, Jr., "Respiratory Exposure to Organic Phosphorus Insecticides," Arch. Environ. Health, 11:564-568 (1965).

Hayes, G. R. Jr., A. J. Funckes, and W. V. Hartwell, "Dermal Exposure of Human Volunteers to Parathion," Arch. Environ. Health, 8:829-833 (1964).

Hayes, W. J., Jr., E. M. Dixon, G. S. Batchelor, and W. M. Upholt, "Exposure to Organic Phosphorus Sprays and Occurrence of Selected Symptoms," Public Health Reports, U.S. Dept. HEW, Public Health Service, 72(9):787-794 (September 1957).

Hayes, W. J., Jr., "Studies on Exposure During the Use of Anticholinesterase Pesticides," Bulletin of the World Health Organization, 44:277-288 (1971).

Hayes, W. J., Jr., 1963 Clinical Handbook on Economic Poisons, PHS Publication No. 476, Washington, D.C. (1963).

Hayes, W. J., Jr., "Toxicity of Pesticides to Man--Risks from Present Levels," Proc. R. Soc. Lond., 167(1007):101-127 (1967).

Hazleton, L. W., and E. G. Holland, "Pharmacology and Toxicology of Parathion," Adv. Chem. Series No. 1, 31:31-38 (1950).

Hitchcock, M., and S. D. Murphy, "Activation of Parathion and Guthion by Tissues of Mammalian, Avian, and Piscine Species," Fed. Proc., 26(2):427 (1967a).

Hitchcock, M., and S. D. Murphy, "Enzymatic Reduction of 0,0-(4-Nitrophenyl) Phosphorothioate, 0,0-Diethyl 0-(4-Nitrophenyl) Phosphate, and 0-Ethyl 0-(4-Nitrophenyl) Benzene Thiophosphonate by Tissues from Mammals, Birds, and Fishes," Biochem. Pharmacol., 16(9):1801-1811 (1967b).

Hollingworth, R. M., R. L. Alstott, and R. D. Litzenberg, "Glutathion S-Aryl Transferase in the Metabolism of Parathion and Its Analogs," Life Sci., 13:191-199 (1973).

Jacobsen, P. L., R. C. Spear, E. Wei, "Parathion and Diisopropylfluorophosphate (DFP) Toxicity in Partially Hepatectomized Rats," Toxicol. Appl. Pharmacol., 26:314-317 (1973).

Jegier, Z., "Health Hazards in Insecticides Spraying of Crops," Arch. Environ. Health, 8:670-674 (1964).

Jensen, J. A., W. F. Durham, and G. W. Pearce, "Studies on Fate of Parathion in Rabbits, Using Radioactive Isotope Techniques," AMA Arch. Ind. Hyg. Occup. Med., 6:326-331 (1952).

Kanagartanam, K., W. H. Boon, and T. K. Hoh, "Parathion Poisoning from Contaminated Barley," Lancet, 1:538-542 (1960).

Khera, K. S., and S. Bedok, "Effects of Thiol Phosphates on Notochordal and Vertebral Morphogenesis in Chick and Duck Embryos," Food Cosmet. Toxicol., 5:359-365 (1967).

Khera, K. S., and D. A. Lyon, "Chick and Duck Embryos in the Evaluation of Pesticide Toxicity," Toxicol. Appl. Pharmacol., 13:1-15 (1968).

Khera, K. S., and D. J. Clegg, "Perinatal Toxicity of Pesticides," Can. Med. Assoc. J., 100:167-172 (1969).

Kimbrough, R. D., and T. B. Gaines, "Effect of Organic Phosphorus Compounds and Alkylating Agents on the Rat Fetus," Arch. Environ. Health, 16:805-808 (1968).

Kimmerle, G., and D. Lorke, "Toxicology of Insecticidal Organophosphates," Pflanz. Nachr. Bayer, 21:111-142 (1968).

Konst, H., and P. J. Plummer, "Acute and Chronic Toxicity of Parathion to Warm-Blooded Animals," Can. J. Comp. Med. Vet. Sci., 14:90-108 (1950).

Lauwerys, R., and J. P. Buchet, "Effect of Two Organophosphorus Esters on Mammary Carcinogenesis by 7,12-Dimethylbenz(α)anthracene," Eur. J. Toxicol., 5:163-167 (1972).

Lewis, W. H., and A. G. Richards, Jr., "Non-Toxicity of DDT on Cells in Cultures," Science, 102:330-331 (1945).

Lichtenstein, E. P., T. W. Fuhremann, A. A. Hochberg, R. N. Zahlten, and F. W. Stratman, "Metabolism of $[^{14}C]$ Parathion and $[^{14}C]$ Paraoxon with Fractions and Subfractions of Rat Liver Cells," J. Agr. Food Chem., 21(3):416-423 (1973).

Lieben, J., R. K. Waldman, and L. Krause, "Urinary Excretion of Paranitrophenol Following Parathion Exposure," AMA Arch. Ind. Hyg. Occup. Med., 6:491-495 (1952).

Litterst, C. L., E. P. Lichtenstein, and K. Kajiwara, "Effects of Insecticides on Growth of HeLa Cells," J. Agr. Food Chem., 17:1199-1203 (1969).

Litterst, C. L., and E. P. Lichtenstein, "Effects and Interactions of Environmental Chemicals on Human Cells in Tissue Culture," Arch. Environ. Health, 22:454-459 (1971).

Lutz, H., and Y. Lutz-Ostertag, "The Action of Different Pesticides on the Development of Bird Embryos," Drugs and Fetal Development, Proc. International Symposium on the Effect of Prolonged Drug Usage on Fetal Development, Beit-Berl, Kfar Saba, Israel, September 1971, pp. 127-150 (1972).

Lutz-Ostertag, Y., and R. Meiniel, "In vitro Sterilizing Effect of Parathion on Embryonic Ovaries of Chick and Quail," C. R. Acad. Sci., Paris, Series D, 267:2178-2180 (1968).

Lutz-Ostertag, Y., R. Meiniel, and H. Lutz, "Action du parathion sur le developpement de l'embryon de caille," C. R. Acad. Sci., Paris, Series D, 268:2911-2913 (1969a).

Lutz-Ostertag, Y., R. Meiniel, and H. Lutz, "Effects du parathion sur le developpement de l'embryon de caille et de certains de ses organes in vivo et in vitro," Biol. Abstr., 52(22):12, 271 (1969b).

Lutz-Ostertag, Y., R. Meiniel, and H. Lutz, "Parathion, Embryonic Development, Sterilization and Estrogenic Effects in Birds; Comparison with the Effects of Aldrin," Anneé Biol., 9:501-507 (1970).

Maibach, H. I., R. J. Feldmann, T. H. Milby, and W. F. Serat, "Regional Variation in Percutaneous Penetration in Man," Arch. Environ. Health, 23:208-211 (1971).

Malkinson, F., "Percutaneous Absorption of Toxic Substances in Industry," AMA Arch. Ind. Health, 21:87-99 (1960).

Marliac, J. P., "Toxicity and Teratogenic Effects of 12 Pesticides in the Chick Embryo," 48th Ann. Meeting Fed. Amer. Soc. Exp. Biol., 105 (1964).

Matsumura, F., and C. T. Ward, "Degradation of Insecticides by the Human and the Rat Liver," Arch. Environ. Health, 13:257-261 (1966).

Medved, L. I., E. I. Spynu, and Iu. S. Kagan, "The Method of Conditioned Reflexes in Toxicology and Its Application for Determining the Toxicity of Small Quantities of Pesticides," Residue Rev., 6:42-74 (1964).

Meiniel, R., Y. Lutz-Ostertag, and H. Lutz, "Teratogenic Effects of Parathion (Organophosphorus Insecticide) on the Skeleton of the Embryo of the Japanese Quail (Coturnix coturnix japonica)," Arch. Anat. Microsc. Morphol. Exp., 59(2):167-183 (1970).

Milby, T. H., and F. Ottoboni, "Report of an Epidemic of Organic Phosphate Poisoning in Peach Pickers, Stanisiaus County, California," State of California Department of Health, Bureau of Occup. Health (1963).

Mol, J. C. M., D. L. Harrison, and R. H. Telfer, "Parathion: Toxicity to Sheep and Persistence on Pasture and in Soil," N. Z. J. Agr. Res., 15:306-320 (1972).

Mrak, E. M., "Report on the Secretary's Commission on Pesticides and Their Relationship to Environmental Health," U.S. Dept. HEW, p. 665 (1969).

Muller, H. D., and D. C. Lockman, "Fecundity and Progeny Growth Following Subacute Insecticide Ingestion by the Mallard," Poul. Sci., 51:239-241 (1972).

Nabb, D. P., W. J. Stein, and W. J. Hayes, Jr., "Rate of Skin Absorption of Parathion and Paraoxon," Arch. Environ. Health, 12:501-505 (1966).

Nakatsugawa, T., and P. A. Dahm, "Microsomal Metabolism of Parathion," Biochem. Pharmacol., 16(1):25-38 (1967).

Nakatsugawa, T., N. M. Tolaman, and P. A. Dahm, "Degradation of Parathion in the Rat," Biochem. Pharmacol., 18(5):1103-1114 (1969).

Namba, T., "Cholinesterase Inhibition by Organophosphorus Compounds and Its Clinical Effects," Bulletin of the World Health Organization, 44:289-307 (1971).

Natoff, I. L., "Influence of the Route of Administration on the Toxicity of Some Cholinesterase Inhibitors," J. Phar. Pharmacol., 19:612-616 (1967).

Natoff, I. L., "Influence of the Route of Exposure on the Acute Toxicity of Cholinesterase Inhibitors," Europ. J. Toxicol., 3:363-367 (1970).

Neal, R. A., "Studies of the Enzymic Mechanism of the Metabolism of Diethyl 4-Nitrophenyl Phosphorothionate (Parathion) by Rat Liver Microsomes," Biochem. J., 105:289-297 (1967a).

Neal, R. A., "Studies on the Metabolism of Diethyl 4-Nitrophenyl Phosphorothionate (Parathion) in vitro," Biochem. J., 103:183-191 (1967b).

Neal, R. A., "A Comparison of the in vitro Metabolism of Parathion in the Lung and Liver of the Rabbit," Toxicol. Appl. Pharmacol., 23:123-130 (1972).

Neill, D. D., H. D. Muller, and J. V. Shutze, "Pesticide Effects on the Fecundity of the Gray Partridge," Bull. Environ. Contam. Toxicol., 6(6):546-551 (1971).

Nevkovic, N., S. Vitorovic, and M. Plesnicar, "The Role of Liver Microsomal Enzymes in the Metabolism of Parathion," Biochem. Pharmacol., 22:2943-2946 (1973).

Norman, B. J., J. A. Roth, and R. A. Neal, "Effect of Temperature on the Mixed Function Oxidase-Catalyzed Metabolism of O,O-Diethyl p-Nitrophenyl Phosphorothionate (Parathion)," Toxicol. Appl. Pharmacol., 26:203-208 (1973a).

Norman, B. J., W. K. Vaughn, and R. A. Neal, "Studies of the Mechanisms of Metabolism of Diethyl p-Nitrophenyl Phosphorothionate (Parathion) by Rabbit Liver Microsomes," Biochem. Pharmacol., 22:1091-1101 (1973b).

O'Brien, R. D., Toxic Phosphorus Esters, New York: Academic Press, pp. 227-228 (1960).

Oga, S., C. C. Pellegatti, S. Reis, and A. C. Zanini, "Toxic and/or Teratogenic Activity of Anticholinesterase Compounds. I. Tetatogenicity of Parathion in Chicken Embryos," Rev. Farm. Bioquim. Univ. Sao Paulo, 9(2):343-355 (1971).

Orzel, R. A., and L. R. Weiss, "The Effect of Various Chemicals on Rat Brain Cholinesterase Inhibition by Parathion," Arch. Int. Pharmacodyn. Thér., 164(1):150-157 (1966).

Osmun, J. W., "PASS Information Relating to Agricultural Jobs," Internal EPA Memo to Ed Johnson (1 April 1974).

Pankaskie, J. E., F. C. Fountaine, and P. A. Dahm, "The Degradation and Detoxification of Parathion in Dairy Cows," J. Econ. Entomol., 45:51-60 (1952).

Phillips, W. E. J., G. Hatina, D. C. Villeneuve, and D. L. Grant, "Effect of Parathion Administration in Rats Following Long-Term Feeding with PCB's," Environ. Physiol. Biochem., 2:165-169 (1972).

Quarles, J., "Worker Protection Standards for Agricultural Pesticides," Federal Register, 39(2):16888-16891 (10 May 1974).

Quinby, G. E., and A. B. Lemmon, "Parathion Residues as a Cause of Poisoning in Crop Workers," J. Amer. Med. Assoc., 166(7):740-746 (1958).

Radeleff, R. D., and G. T. Woodard, "The Toxicity of Organic Phosphorus Insecticides to Livestock," J. Am. Vet. Med. Assoc., 130:215-216 (1957).

Radeleff, R. D., G. T. Woodard, W. J. Nickerson, and R. C. Bushland, "Part II. Organic Phosphorus Insecticides," The Acute Toxicity of Chlorinated Hydrocarbon and Organic Phosphorus Insecticides to Livestock, U.S. Dep. of Agriculture, Tech. Bull. 1122, pp. 35-46 (1955).

Radeleff, R. D., "The Toxicity of Insecticides and Herbicides to Livestock," Adv. Vet. Sci., 4:265-276 (1958).

Radeleff, R. D., and R. C. Bushland, "The Toxicity of Pesticides for Livestock, "Symposium: The Nature and Fate of Chemicals Applied to Soils, Plants, and Animals," Agricultural Research Service, U.S. Dep. of Agriculture.

Rider, J. A., and H. C. Moeller, "Tolerance of Organic Phosphates in Man," Progress Report of Franklin Hospital Foundation, San Francisco, California (1 October 1963).

Rider, J. A., and H. C. Moeller, "Studies on the Anticholinesterase Effects of Systox and Methyl Parathion in Humans," Fed. Proc., 23(2): 176 (1964).

Rider, J. A., H. C. Moeller, and E. J. Puletti, "Continuing Studies on Anticholinesterase Effect of Methyl Parathion, Initial Studies with Guthion, and Dichlorvos on Humans," Fed. Proc., 26(2):427 (1967).

Rider, J. A., H. C. Moeller, E. J. Puletti, and J. I. Swader, "Toxicity of Parathion, Systox, Octamethyl Pyrophosphoramide, and Methyl Parathion in Man," Toxicol. Appl. Pharmacol., 14(3):603-611 (1969a).

Rider, J. A., and E. J. Puletti, "Studies on the Anticholinesterase Effects of Gardona, Methyl Parathion, and Guthion in Human Subjects," Fed. Proc., 28(2):479 (1969b).

Roan, C. C., D. P. Morgan, N. Cook, and E. H. Paschal, "Blood Cholinesterases, Serum Parathion Concentrates and Urine p-Nitrophenol Concentrations in Exposed Individuals," Bull. Environ. Contam. Toxicol., 4(6):362-369 (1969).

Roger, J-C., D. G. Upshall, and J. E. Casida, "Structure-Activity and Metabolism Studies on Organophosphate Teratogens and Their Alleviating Agents in Developing Hen Eggs with Special Emphasis on Bidrin," Biochem. Pharmacol., 18(2):373-392 (February 1969).

Rosival, L., F. V. Selecky, and L. Vbrovsky, "Acute Experimental Poisoning with Organophosphorus Insecticides," Bratisl. Lek. Listy, 38:151-160 (1958).

Roudabush, R. L., C. J. Terhaar, D. W. Fassett, and S. P. Dziuba, "Comparative Acute Effects of Some Chemicals on the Skin of Rabbits and Guinea Pigs," Toxicol. Appl. Pharmacol., 7:559-565 (1965).

Sakai, K., and F. Matsumura, "Degradation of Certain Organophosphate and Carbamate Insecticides by Human Brain Esterases," Toxicol. Appl. Pharmacol., 19(4):660-666 (1971).

Schaefer, R. A., and G. M. Vance, "Exposure of Connecticut Tobacco Workers to Parathion," AMA Arch. Ind. Hyg. Occup. Med., 7:193-196 (1953).

Shellenberger, T. E., G. W. Newell, R. F. Adams, and J. Barbaccia, "Cholinesterase Inhibition and Toxicologic Evaluation of Two Organophosphate Pesticides in Japanese Quail," Toxicol. Appl. Pharmacol., 8:22-28 (1966).

Shishido, T., and J. Fukami, "Studies on the Selective Toxicites of Organic Phosphorus Insecticides (II)," Botyu-Kagaku, 28(1):69-76 (February 1963).

Spynu, E. I., "Data Concerning the Toxicology of the Insecticide Niuiph 100 and the Establishment of Tolerance Limits," Dissertation, Kiev (1952).

Spynu, E. I., "The Effect of Some Organophosphorus Insecticides on the Higher Nervous Activities and on the Cholinesterase Activity," The Chemistry and Application of Organophosphorus Compounds, Acad. Sci. USSR (ed.), Moscow (1957).

Stevens, J. T., "The Effect of Parathion on the Metabolism of ^{3}H-Testosterone by Hepatic Microsomal Enzymes from the Male Mouse," Pharmacology, 10(4):220-225 (1973).

Sumerford, W. T., W. J. Hayes, Jr., J. M. Johnston, K. Walker, and J. Spillane, "Cholinesterase Response and Symptomatology from Exposure to Organic Phosphorus Insecticides," AMA Arch. Ind. Hyg. Occup. Med., 7:383-398 (1953).

Taylor, W. J., and Z. N. Gaut, "Poisoning with the Newer Organophosphorus Insecticides," Int. J. Clin. Pharmacol., 1(3):175-183 (1968).

Technical Development Laboratories, Communicable Disease Center, Clinical Memoranda on Economic Poisons, U.S. Dept. HEW, Public Health Service, pp. 7-12 and 17-21 (1 April 1956).

Triolo, A. J., and J. M. Coon, "Toxicologic Interactions of Chlorinated Hydrocarbon and Organophosphate Insecticides," J. Agr. Food Chem., 14(6):549-555 (1966).

Tsachalinas, D., G. Logaras, and A. Paradelis, "Observations on 246 Cases of Acute Poisoning with Parathion in Greece," Eur. J. Toxicol., 4:46-49 (1971).

Villeneuve, D. C., W. E. J. Phillips, and J. Syrotiuk, "Modification of Microsomal Enzyme Activity and Parathion Toxicity in Rats," Bull. Environ. Contam. Toxicol., 5(2):125-132 (1970).

Villeneuve, D. C., and W. E. J. Phillips, "The Effect of Acute Ethanol Administration on Parathion Toxicity and in vitro Parathion Degradation on the Rat," Can. J. Physiol. Pharmacol., 49:481-483 (1971).

Villeneuve, D. C., R. F. Willes, J. B. Lacroix, and W. E. J. Phillips, "Placental Transfer of ^{14}C-Parathion Administered Intravenously to Sheep," Toxicol. Appl. Pharmacol., 21:542-548 (1972).

Vukovich, R. A., A. J. Triolo, and J. M. Coon, "The Effect of Chlorpromazine on the Toxicity and Biotransformation of Parathion in Mice," J. Pharmacol. Exp. Thera., 178(2):395-401 (1971).

Ware, G. W., D. P. Morgan, B. J. Estesen, W. P. Cahill, and D. M. Whitacre, "Establishment of Reentry Intervals for Organophosphate-Treated Cotton Fields Based on Human Data: I. Ethyl and Methyl Parathion," Arch. Environ. Contam. Toxicol., 1:48-59 (1973).

Webb, R. E., C. C. Bloomer, and C. L. Miranda, "Effect of Casein Diets on the Toxicity of Malathion and Parathion and Their Oxygen Analogues," Bull. Environ. Contam. Toxicol., 9(2):102-107 (1973).

Weiss, L. R., J. Bryant, and O. G. Fitzhugh, "Blood Sugar Levels Following Acute Poisoning with Parathion and 1-Naphthyl N-Methyl Carbamate (Sevin) in Three Species," Toxicol. Appl. Pharmacol., 6(3):363 (1964).

Weiss, L. R., and R. A. Orzel, "Enhancement of Toxicity of Anticholinesterases by Central Depressant Drugs in Rats," Toxicol. Appl. Pharmacol., 10:334-339 (1967).

Westerman, E., "Accumulation of Environmental Agents or Their Effects in the Body," Environ. Res., 2:340-351 (1969).

Wilber, C. G., and R. A. Morrison, "The Physiological Action of Parathion in Goats," Am. J. Vet. Res., 10(59):308-313 (1955).

Williams, C. H., "β-Glucuronidase Activity in the Serum and Liver of Rats Treated with Parathion," Toxicol. Appl. Pharmacol., 16:533-539 (1970).

Williams, J. W., and J. T. Griffiths, "Parathion Poisoning in Florida Citrus Spray Operations," J. Fla. Med. Assoc., 37:707-709 (1951).

Wolfe, H. R., W. F. Durham, and J. F. Armstrong, "Exposure of Workers to Pesticides," Arch. Environ. Health, 14:622-633 (1967).

Wolfe, H. R., W. F. Durham, and J. F. Armstrong, "Urinary Excretion on Insecticide Metabolites," Arch. Environ. Health, 21:711-716 (1970).

Yamada, A., "Teratogenic Effects of Organophosphorus Insecticides in the Chick Embryo," Osaka Shiritsu Daigaku Igaku Zasshi, 21:245-255 (1972).

SUBPART II. C. FATE AND SIGNIFICANCE IN THE ENVIRONMENT

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Effects on Aquatic Organisms

Fish -

Laboratory studies - Acute toxicity data for parathion's effect on fish is summarized in Table 18. It is obvious that toxicity of parathion varies considerably depending upon the species (see Table 19 for scientific names of fish) and upon the method used. The toxicities of parathion to the natural population of fish ranges from a low of 0.047 ppm (48 hr LC$_{50}$) in bluegill to a high of 6.25 ppm (48 hr TL$_m$) in carp. Exposure times used in the studies cited in Table 18 ranged from 2 hr to 1 week. Some of the differences in toxicity are undoubtedly due to differences in the maturity of the fish used and in their physical condition.

The effects of maturity on the toxicity of parathion for bluegill can be inferred from the TL$_m$ data in Table 20. There is a difference in the susceptibility of young (small) bluegill compared to older (large) bluegill; the larger fish are less susceptible to parathion poisoning. While the difference in TL$_m$ is not great (about two times), the contamination of a water system by parathion at a level below the TL$_m$ for adult fish could still have a lethal effect on the immature members of the population.

Pickering et al. (1962)[1] have made a comparison of the toxicity of organophosphate insecticides for bluegill and white rats (Table 21). It would be more valid to compare fish toxicity, which is associated with continuous contact with the pesticide through the gills, with inhalation data for rats, which are derived through continuous exposure to the respiratory tract. Kimmerle and Lorke (1968)[2] reported an inhalation LC$_{50}$ for male rats to be 0.0315 mg/liter with a 4 hr exposure time.

The reduction in fish-brain acetylcholinesterase (AChE) activity may be used to monitor waters for the presence of organophosphate contaminants although cautious interpretations should be made (Gibson et al. 1969)[3] Nevertheless, Coppage and Matthews (1974)[4] state: "...AChE measurements are probably the best general index of organophosphate poisoning of fish in the environment."

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[1] Pickering, Q. H., C. Henderson, and A. E. Lemke, "The Toxicity of Organic Phosphorus Insecticides to Different Species of Warmwater Fishes," Trans. Am. Fish. Soc., 91(2):1750184 (1962).

[2] Kimmerle, G., and D. Lorke, "Toxicology of Insecticidal Organophosphates," Pflanz. Nachr. Bayer. 21:111-142 (1968).

[3] Gibson, J. R., J. L. Ludke, and D. E. Ferguson, "Sources of Error in the Use of Fish-Brain Acetylcholinesterase Activity as a Monitor for Pollution," Bull. Environ. Contam. Toxicol., 4:17-23 (1969).

[4] Coppage, D. L., and E. Matthews, "Short-Term Effects of Organophosphate Pesticides on Cholinesterases of Estuarine Fishes and Pink Shrimp," Bull. Environ. Contam. Toxicol., 11:483-488 (1974).

Table 18. ACUTE TOXICITY OF PARATHION TO FISH

Fish tested	Exposure time (hr)	Toxicity calculated (oral)	Toxicity measured (ppm)	References
Fathead minnow	96	TL_m	1.4-1.6	a/, b/
Fathead minnow	96	TL_{50}	2.5	c/
Bluegill	96	TL_m	0.095	d/
Bluegill	48	LC_{50}*	0.047	e/
Bluegill	24	LC_{50}	0.141	d/
Carp	48	TL_m	3.2-6.25	f/, g/
Carp	24	40% Mortality	1.00	h/
Rainbow trout	48	80% Mortality	2.00	i/
Rainbow trout	1 Week	50% Mortality	1.30	j/
Brook trout	96	TL_{50}	1.80	c/
Pumpkinseed sunfish	96	TL_m	0.155-0.42	a/, d/
Pumpkinseed sunfish	24	LD_{50}	110 mg/kg	k/
Green sunfish	24	LC_{50}	0.155	d/
Largemouth bass	96	TL_m	0.19	a/
Mummichog	48	EC_{50}*	0.15	l/
Tilapia	96	LC_{50}	0.375	m/
Tilapia	96	TL_m	0.60	f/
Striped mullet	96	LC_{50}	0.125	m/
Golden shiner	24	LC_{50}	0.931	d/
Bullhead	2	IP-0 Mortality	40 mg/kg	n/
Mosquito fish	36	50% Mortality	0.19	i/
Rice fish	48	TL_m	2.9	g/
Goldfish	96	TL_m	2.7	a/
Guppies	96	TL_m	0.06	a/

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* Because death was used as the end point, LC_{50}, EC_{50}, and TL_m values are comparable.

a/ Pickering et al., *op. cit.* (1962).

b/ Henderson, C., and Q. H. Pickering, "Toxicity of Organic Phosphorus Insecticides to Fish," *Trans. Am. Fish.Soc.*, 87:39-51 (1958).

c/ Anon., National Water Quality Laboratory (EPA), unpublished data (24 May 1974).

d/ Gibson, J. R., "Comparative Biochemistry of Parathion Metabolism in Three Species of Fishes," *Diss. Abstr. Int.*, 32(4):2365-B (1971).

e/ Federal Water Pollution Control Administration, "Water Quality Criteria," *Report of the National Technical Advisory Committee*, p. 37 (1968).

f/ Sreenivasan, A., and G. K. Swaminathan, "Toxicity of Six Organophosphorus Insecticides to Fish," *Curr. Sci.*, India, 36:397-398 (August 1967).

g/ Nishiuchi, Y., and Y. Hashimoto, "Toxicity of Pesticides to Some Freshwater Organisms," *Rev. Plant Protec. Res.*, 2:137-139 (1969).

h/ Mulla, M. S., J. St. Amant, and L. D. Anderson, "Evaluation of Organic Pesticides for Possible Use as Fish Toxicants," *Prog. Fish-Cult.*, 29(1):36-42 (1967).

i/ Lewallen, L. L., and W. H. Wilder, "Toxicity of Certain Organophosphorus and Carbamate Insecticides to Rainbow Trout," *Mosquito News*, 22(4):369-372 (1962).

j/ Wood, E. M., "The Pathology of Pesticide Toxicity in Fish," Unpublished, USDI, Columbia, Missouri.

k/ Benke, G. M., K. L. Cheever, F. E. Mirer, and S. D. Murphy, "Comparative Toxicity, Anticholinesterase Action and Metabolism of Methyl Parathion in Sunfish and Mice," *Toxicol. Appl. Pharmacol.*, 28:97-109 (1974).

l/ Lowe, J. I., P. D. Wilson, and R. B. Davison, "Laboratory Bioassays," *Progress Report for Fiscal Year 1969*, Bureau of Commercial Fisheries Center for Estuarine and Menhaden Research, Pesticides Field Station, Gulf Breeze, Florida, U.S. Dept. Interior, Circular 335, pp. 20-28 (1970).

m/ Lahav, M., and S. Sarig, "Sensitivity of Pond Fish to Cotnion (Azinphosmethyl) and Parathion," *Bamidgeh*, 21(3):67-74 (1970).

n/ Murphy, S. D., R. R. Lauwerys, and K. L. Cheever, "Comparative Anticholinesterase Action of Organophosphorus Insecticides in Vertebrates," *Toxicol. Appl. Pharmacol.*, 12:22-35 (1968).

Table 19. COMMON AND SCIENTIFIC NAMES OF FISH USED IN CONTROLLED TOXICITY TESTS WITH PARATHION

Common name	Scientific name
Fathead minnow	*Pimephales promelas*
Bluegill	*Lepomis macrochirus*
Carp	*Cyprinus carpio*
Rainbow trout	*Salmo gairdneri*
Brook trout	*Salvelinus fontinalis*
Green sunfish	*Lepomis cyanellus*
Pumpkinseed sunfish	*Lepomis gibbosus*
Largemouth bass	*Micropterus salmoides*
Mummichog	*Fundulus heteroclitus*
Tilapia	*Tilapia aurea*
Striped mullet	*Mugil cephalus*
Golden shiner	*Notemigonus crysoleucas*
Black bullhead	*Ictalurus melas*
Mosquito fish	*Gambusia affinis*
Rice fish	*Oryzias latipes*
Goldfish	*Carassius auratus*
Guppy	*Lebistes reticulatus*
Spot	*Leiostomus xanthurus*
Pinfish	*Lagodon rhomboides*

Table 20. COMPARATIVE TOXICITY OF PARATHION AND MALATHION INSECTICIDES TO CENTRARCHIDS

	96 hr TL_m (ppm, AI)			
Insecticide	Bluegills small	Bluegills large	Green sunfish	Largemouth bass
Parathion	0.065	0.14	0.42	0.19
Malathion	0.11	0.24	0.12	0.05

Data from: Pickering et al., *op. cit.* (1962).

Table 21. TOXICITY OF THREE ORGANOPHOSPHATE INSECTICIDES TO BLUEGILL AND WHITE RATS

Insecticide	Bluegill 96 hr TL_m (mg/1)	White rats Oral LD_{50} (mg/Kg)
Parathion	0.095	13.0
Methyl Parathion	1.90	28.0
Malathion	0.090	1,375.0

Data from: Pickering et al., op. cit. (1962).

The relationship between AChE inhibition to death of estuarine animals was studied by Coppage and Matthew (1974). In this study the effect of parathion on spot and pinfish AChE activity at concentrations of LC_{40} to LC_{60} were determined. When spot were exposed to parathion at 10 ppb for 24 hr, AChE activity (brain) was reduced 88% and when pinfish were used as the test animal the activity was reduced 90%.

These studies appeared to indicate that AChE activity reduced to 80% of normal was critical in short-term organophosphate poisonings. The lethal threshold probably varies among species.

The relationship of brain AChE inhibition to death after exposure to parathion concentrations that killed from 40 60% of sheepshead minnows in 2, 24, 48, and 72 hr was studied by Coppage (1972).[1] The AChE activity was measured at 2 hr for the 2-hr lethal dose, at 6 and 24 hr for the 24-hr lethal dose, at 24 and 48 hr for the 48-hr lethal dose and at 24, 48 and 72 hr for the 72-hr lethal dose.

The 48- and 72-hr exposures caused AChE to fall below 17.7% of normal for 24 hr preceding death. The 24-hr exposure caused AChE to fall to and remain below 17.7% for 18 hr prior to death. Death in all lethal exposures occurred at AChE levels ranging from 0 to 15.4% of normal.

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[1] Coppage, D.L., "Organophosphate Pesticides: Specific Level of Brain AChE Inhibition Related to Death in Sheepshead Minnows," Trans. Am. Fish Soc., 101:534-536 (1972).

Problems that may arise by using in vitro reduction of AChE as a measure of insecticide toxicity were demonstrated by Coppage (1971).[1] He reported that by comparing the percentage of the inhibition of AChE with the 48-hr LD_{50} of parathion and other organophosphates, good correlation between the in vitro inhibition and toxicity could not be established. This author stated that only in vivo inhibition can be a meaningful indicator of toxicity and that this required bioassays in order to determine the exact relationship of AChE inhibition to concentration of insecticide, length of exposure and death.

The responses of mosquito fish which had developed a resistance to organochlorine compounds were compared to those of naturally sensitive fish when both groups were exposed to parathion (Chambers and Yarbrough, 1974).[2] It was shown that organochlorine-resistant populations of mosquito fish can tolerate more parathion in the spring than in the fall (about 1.6-fold greater).

The poisoning effects of parathion in fish usually appear first as erractic movements followed by the fish swimming on its side. As the poisoning progresses, the fish will come to the surface and gulp for air. In most studies the cessation of gill movement is taken to be the time of death.

Parathion poisoning produced lesions in the liver, kidney and gills. However, the liver changes were nonspecific, generalized degenerative changes. Characteristic liver changes in moribund fish were swelling of parenchymal cells and congestion of the sinusoids. The kidney lesions were apparently glomerular damage with the appearance of proteinaceous material both in Bowman's space and in the connecting tubules. Pathology of the gill consisted primarily of thickening of the epithelial layer surrounding the capillaries of the individual lamellae.[3]

Parathion poisoning in the frog is reported to produce anemia and leucopenia. Increases in concentration resulted in a progressive neutropenia and a lymphocystosis. Clotting time was increased.

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[1] Coppage, D. L., "Characterization of Fish Brain Acetylcholinesterase with an Automated pH Stat for Inhibition Studies," Bull. Environ. Contam. Toxicol., 6:304-310 (1971).

[2] Chambers, J. E., and J. D. Yarbrough, "Parathion and Methyl Parathion Toxicity to Insecticide-Resistant and Susceptible Mosquito Fish (Gambusia affinis)," Bull. Environ. Contam. Toxicol., 4:315-320 (1974).

[3] Wood, E. M. "The Pathology Of Pesticide Toxicity in Fish," Unpublished, U.S. Department of Interior, Columbia, Missouri.

Generally toxicosis was shown by a drooping posture and a decreased muscle tone; occasionally spastic paralysis occurred.

When parathion exposure was increased to 15 ppm, a generalized edema resulted. Tremors, salivation or hemorrhage was not observed (Kaplan, and Glaczenski, 1965).[1]

Sanders (1970)[2] reported TL_{50} values in mg/liter for 1-week-old frog tadpoles (*Pseudacris triseriata*) for 24, 48, and 96 hr to be 1.6 (0.4-3.0), 1.4 (0.91-2.8) and 1.0 (0.3-2.0), respectively.

Field studies - Dolphin and Peterson (1960)[3] investigated the fish toxicity of parathion with a view to its possible use for the control of the Clear Lake gnat (*Chaoborus astictopus*) in Clear Lake, Lake County, California. Parathion was added to Clear Lake water at different concentrations in 5-gal aquaria to which five test fish each were added. The 24-hr LC_{50} of parathion to Clear Lake bluegill (*Lepomis machochirus*) was found to be 0.013 ppm. The approximate LC_{90} of parathion to the Clear Lake gnat, the target insect, was 0.018 ppm. Thus, parathion was toxic to fish at dosages required for insect control.

In the summer and fall of 1959, two farm ponds were treated with parathion by injecting a 1:10 acetone-pond water formulation containing the required amount of technical grade parathion from a Hudson pack sprayer into the wake of a small boat which was being rowed back and forth along the length of the pond.

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[1] Kaplan, H. M., and S. S. Glaczenski, "Hematological Effects of Organophosphate Insecticides in the Frog (*Rana pipiens*)," *Life Sci.*, 4(7):1213-1219 (1965).

[2] Sanders, H. O., "Pesticide Toxicities to Tadpoles of the Western Chorus Frog *Pseudacris triseriata* and Fowler's Toad *Bufo woodhousii fowleri*, *Copeia*, No. 2, pp. 246-251 (1970).

[3] Dolphin, R. E., and R. N. Peterson, "Developments in the Research and Control Program of the Clear Lake Gnat *Chaoborus astictopus*, D.&S.," *Proc. and Papers of Twenty-Eighth Ann. Conf.*, California Mosquito Control Assoc., pp. 90-94 (1960).

One pond was treated with parathion at a dilution rate of 1:60 million (0.017 ppm). Four days after treatment, 100% mortality of Clear Lake gnats was achieved. There was heavy fish mortality 2 weeks after treatment, including 388 green sunfish, 11 bluegill, and 12 brown bullhead.

A second farm pond was treated in the same manner with parathion at the rate of 1:100 million (0.01 ppm). In this case, Clear Lake gnat mortality 4 days after treatment was 45%. Two weeks after treatment, four dead bluegill were observed. Thus, the results of the pond tests were in good agreement with the aquarium tests.

Nicholson et al. (1962)[1] and Grzenda et al. (1962)[2] described the results of insecticide contamination of a 2.7-acre pond draining a 40-acre watershed where peaches were the principal crop. Parathion was used for pest control in the watershed from 11 April to 1 August 1960. Before insecticide use started in 1960, parathion residues of 1.7 ppm were present in a surface soil sample, indicating carry-over of parathion residues in the soil from the preceding season. Surface soil samples taken during and after the parathion spray season in 1960 contained from 0.23 to 1.39 ppm of parathion. Prior to insecticide use in 1960, pond bottom mud samples contained 1.9 ppm of parathion, while two water samples contained 0.02 ppb. Most of the parathion present in the pond immediately before the 1960 spray season apparently entered the pond adsorbed on soil during a period of accelerated soil erosion in March of 1960.

The parathion content of the pond water increased as the spraying season progressed, while the parathion content of pond mud decreased, presumably due to decomposition of existing residues and slower soil erosion. Populations of fish, zooplankton, aquatic insects and Oligochaeta were unaffected by the parathion residues in the pond water and mud. However, there was a significant reduction in the numbers of immature aquatic insects. Bluegills that normally feed primarily on immature chironomids fed almost entirely on planktonic Crustacea when the insect crop became severely reduced.

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[1] Nicholson, H. P., H. J. Webb, G. J. Lauer, R. E. O'Brien, A. R. Grzenda, and D. W. Shanklin, "Insecticide Contamination in a Farm Pond. Part I - Origin and Duration," Trans. Am. Fish.Soc., 91(2): 213-221 (1962).

[2] Grzenda, A. R., G. J. Lauer, and H. P. Nicholson, "Insecticide Contamination in a Farm Pond Part II - Biological Effects," Trans. Am. Fish.Soc., 91(2):213-221 (1962).

The amounts of parathion found in the pond water (0.01 to 1.22 ppb) were well below known levels of acute toxicity to fish. The study demonstrates that bluegill populations can survive chronic exposure to parathion in the range of 0.01 to 1.22 ppb for at least 6 months without detectable adverse effects. The fish in this field study may actually have been exposed more or less continuously to low levels of parathion for at least 17 months.

Mulla et al. (1963)[1/] investigated the toxicity of parathion and other insecticides to some aquatic wildlife species including the mosquito fish (Gambusia affinis) and the aquatic larval stages of the bullfrog (Rana catesbeiana), the Western toad (Bufo boreas) and Hammond's spadefoot toad (Scaphiopus hammondi). The toxicants were applied to 1/16-acre field ponds at a volume of 8 gal/acre as aqueous sprays prepared from emulsion concentrates. At the rate of 0.1 lb AI/acre, parathion produced 18% mortality of mosquito fish 1 day after treatment. At the rate of 0.4 lb AI/acre, mosquito fish mortality was 96% when the fish were exposed 1 day after treatment, 32% when exposed after 2 days, and 8% when exposed after 3 days. The same application rates of parathion were nontoxic to the tadpoles when these were exposed to the contaminated water for 24 hr.

Miller et al. (1966)[2/] studied the water translocation of parathion and its subsequent occurrence in fish and mussels in a simulated cranberry bog in the greenhouse. ^{35}S-labeled parathion was applied at the rate of 1 lb AI/acre to the model bog. The bog was flooded 24 hr after application, and the water was left on for 4 hr, simulating a frost protection flooding. Estuarine fish (Fundulus heteroclitus) and freshwater mussels (Elliptio complanatus) were then exposed to the contaminated water in aquaria. Specimens were analyzed periodically.

The majority of the parathion originally present disappeared from the water within 144 hr. During this time, three labeled parathion degradation products were encountered. Of 60 fish present in the aquarium, only 12 were alive after the first 24 hr. The parathion concentration in the aquarium declined from 0.07 to 0.02 ppm during this period. After 48 hr, three of the remaining fish showed symptoms of poisoning.

1/ Mulla, M. S., L. W. Isaak, and H. Axelrod, "Field Studies on the Effects of Insecticides on Some Aquatic Wildlife Species," J. Econ. Entomol., 56(2):184-188 (1963).

2/ Miller, C. W., B. M. Zuckerman, and A. J. Charig, "Water Translocation of Diazinon-^{14}C and Parathion-^{35}S Off a Model Cranberry Bog and Subsequent Occurrence in Fish and Mussels," Trans. Am. Fish. Soc., 95:345-349 (1966).

On analysis, these specimens showed an accumulation of parathion to 2.1 ppm, while the surrounding aquarium water at this time analyzed 0.01 ppm parathion. Mussels also accumulated parathion, but to a lesser degree than the fish.

Holland et al. (1967)[1/] employed a fish brain acetylcholinesterase test to monitor pollution by organophosphorus pesticides along the Atlantic and Gulf coasts. Seven laboratories cooperated by sending fish from their areas periodically for AChE assay. A total of 93 samples of spot (Leiostomus xanthurus) and sheepshead minnows (Cyprinodon variegatus) from 43 stations were analyzed. Low AChE activity was found in 17 samples (18.3%), but 13 of these were from only two areas. Fish that showed low AChE activity (73 to 88% of normal) from South Carolina came from the vicinity of the Ashley River near Charleston. This river receives wastes from plants producing a variety of organophosphate chemicals. Fish from the Galveston area with low enzyme activity (75 to 84% of normal) generally came from stations along the eastern edge of Trinity Bay. The authors did not ascertain which organophosphate insecticides may have been responsible for the observed fish brain AChE depression.

The Environmental Protection Agency's Gulf Breeze Environmental Research Laboratory recently analyzed samples of whole fish and of fish organs collected by the Texas Parks and Wildlife Department at different points in Texas for residues of parathion and other pesticides. Parathion residues found ranged from 21 to 170 ppb, wet weight. The parathion residue levels were generally much lower than residues of organochlorine pesticides found in the same specimens.

The Water Quality Criteria Data Book, Vol 3 (Battelle Columbus, 1971),[2/] a summary review of the effects of commercial chemical products on aquatic organisms, includes references to two further reports on parathion fish toxicity. When parathion was applied for the control of mosquito larvae to a lake in Salt Lake County, Utah, at the rate of 0.35 lb AI/acre, there were no fish losses, and no adverse effects were observed on mammals, birds, reptiles, and amphibians. Invertebrates were not affected uniformly. Crustaceans and larvae of the insect family Ephydridae were not harmed, but spiders and aquatic insects other than Ephydridae were harmed in various degrees. Aquatic beetles were affected more seriously than other insects, except mosquito larvae.

[1/] Holland, H. T., D. L. Coppage, and P. A. Butler, "Use of Fish Brain Acetylcholinesterase to Monitor Pollution by Organophosphorus Pesticides," Bull. Environ. Contam. Toxicol., 2(3):156-162 (1967).

[2/] Battelle's Columbus Laboratories, "Effects of Chemicals on Aquatic Life," Water Quality Criteria Data Book, Vol. 3:B167-B186, Environmental Protection Agency (1971).

Mulla and Isaak (1961)[1] treated ponds in the Bakersfield, California, area with parathion from a 50% emulsifiable concentrate. At 0.1 lb AI/acre, 22% mortality of mosquito fish (Gambusia affinis) occurred in 24 hr. At 0.4 lb parathion AI/acre, there was 92% mortality of the fish in 24 hr. In these field tests, the fish were exposed in cages placed in the ponds.

The Federal Water Pollution Control Administration's "Water Quality Criteria," (1968) includes parathion in "Pesticide Group A," defined as chemicals that are acutely toxic to shrimp at concentrations of 5 µg/liter and less. On the assumption that 1/100 of this level represents a reasonable safety factor, it is recommended that environmental levels of these substances not be permitted to rise above 50 ng/liter of water. This level is so low that the pesticides in this category (including parathion) could not be applied directly in or near the marine habitat without danger of causing damage. The 48-hr TL_m of parathion to shrimp is listed at 1.0 µg/liter. Shrimp were selected as the test organism because they are among the most sensitive marine organisms in regard to their reaction to chemicals.

Lower Aquatic Organisms - For purposes of this review, "lower aquatic organisms" are defined to include primary producers (phytoplankton, attached algae, moss, and vascular plants); consumers (protozoa, rotifers, and crustacea); benthic invertebrates (annelids, insects, crustacea, and mollusca); decomposers (fungi and bacteria).

Laboratory studies - The aquatic species other than fish and frogs which have been studied under controlled conditions are few in number. Lowe et al. (1970) studied the effect of parathion on shrimp (Penaeus duorarum), and oysters (Crassostrea virginica). They found that a 48-hr EC_{50} was obtained with juvenile shrimp at a parathion concentration of 0.0002 ppm, and that oyster shell growth was decreased 22 percent at 1.0 ppm in a 96-hr test. Naqvi and Ferguson (1970)[2] found that the LD_{50} (24 hr) for freshwater shrimp (Palaemonetes kadiakensis) was 0.0082 ppm.

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[1] Mulla, M. S., and L. W. Isaak, "Field Studies on the Toxicity of Insecticides to the Mosquito Fish, Gambusia affinis," J. Econ. Entomol., 54(6):1237-1242 (1961).

[2] Naqvi, S. M., and D. E. Ferguson, "Levels of Insecticides Resistance in Freshwater Shrimp, Palaemonetes kadiakensis," Trans. Am. Fish. Soc., 99:697 (1970).

These data also indicate that parathion contamination of streams and other aquatic habitats at levels well below that having a direct toxic effect on fish can still have an indirect effect by destroying organisms in the food chain.

Lowe et al. (1971)[1] performed a study in which oysters were grown from juvenile to sexual maturity in seawater polluted with 1 ppb parathion. A significant difference between the weights of treated and of the untreated controls was not found.

Field studies - The Federal Water Pollution Control Administration's "Water Quality Criteria" (1968) lists the 48-hr TL_m values of parathion to the stonefly (Pteronarcys californica) and to Daphnia pulex as 11 and 0.4 µg/liter, respectively. Parathion ranks intermediate among the insecticides included in this listing in regard to its toxicity to these two aquatic organisms.

Further data on the toxicity of parathion to Daphnia pulex and to other zooplankton organisms, and to benthic invertebrates are summarized in Tables 22 and 23. The immobilization values (EC_{50}) of parathion to the zooplankton species range from 0.37 to 0.8 ppb. The LC_{50} values of parathion to several species of stoneflies, caddisflies and mayflies, and to one species each of amphipods and freshwater shrimp varied considerably, depending upon the species and the experimental conditions such as temperature and length of exposure. Parathion was considerably more toxic to most of these species than, for instance, malathion. However, it was less toxic than malathion to the amphipod (Gammarus lacustris).

Ware and Roan (1971)[2] reviewed the literature on the interaction of pesticides with aquatic microorganism and plankton. The data cited from several original research papers in a brief section dealing with organophosphate insecticides again confirm that parathion, along with other organic phosphates, is subject to adsorption and degradation by such organisms as yeasts (Torulopsis utilis), bacteria (Pseudomonas fluorescens, Thiobacillus thiooxidans, Bacillus subtilis), and green algae (Chlorella pyrenoidosa).

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[1] Lowe, J. I., P. D. Wilson, and A. J. Wilson, "Chronic Exposure of Oysters to DDT, Toxaphene and Parathion," Proc. Nat. Shellfish Assoc., 61:71-79 (1971).

[2] Ware, G. W., and C. C. Roan, "Interaction of Pesticides with Aquatic Microorganisms and Plankton," Residue Reviews, 33:15-45 (1971).

Table 22. EC_{50} (IMMOBILIZATION) VALUES (ppb) OF THREE ORGANOPHOSPHATE INSECTICIDES TO ZOOPLANKTON

Species	Temperature (°F)	Time (hr)	Malathion	Parathion	Methyl parathion	Reference
Daphnia pulex	21°C	48	2			a/
	--	48		0.76	LC_{50}	b/
	60	48	1.8	0.6		c/
	60	48	1.8			d/
Daphnia magna	68	24	0.9	0.8		c/
	68	48			4.8	d/
	68	50	0.9	0.8		c/
	20°C	50	0.9	0.8		e/
Daphnia carinata	78	64	0.2	0.5		f/
Simocephalus serrulatus	60	48	3.5	0.37		c/
	70	48	6.2	0.47		c/

Source: Li, M., and R. A. Fleck, "The Effects of Agricultural Pesticides in the Aquatic Environment, Irrigated Croplands, San Joaquin Valley," Pesticide Study Series - 6, Environmental Protection Agency, Office of Water Programs, Applied Technology Division, Rural Waste Branch TS-00-72-05, 268 pp. (1972).

a/ Cope, O. B., "Contamination of the Freshwater Ecosystem by Pesticides," *J. Appl. Ecol.*, 3 (Suppl):33-44 (A special issue on Pesticides in the Environment and Their Effects on Wildlife) (1966). (In: Li and Fleck, 1972.)

b/ Priester, L. E., "The Accumulation in Metabolism of DDT, Parathion and Endrin by Aquatic Food-Chain Organisms," Ph.D. Thesis, Clemson University (1965). (In: Li and Fleck, 1972.)

c/ Sanders, H. O., and O. B. Cope, "Toxicities of Several Pesticides to Two Species of Cladocerans," *Trans. Am. Fish. Soc.*, 95(2):165-169 (1966).

d/ Federal Water Pollution Control Administration, *Water Quality Criteria*, Report of the National Technical Advisory Committee, p. 37 (1968).

e/ Anderson, B. G., "The Toxicity of Organic Insecticides to *Daphnia*." In: *Transactions of the Second Seminar on Biological Problems in Water Pollution*, Cincinnati, Ohio, U.S. Public Health Service, pp. 94-95 (1959). (In: Li and Fleck, 1972.)

f/ Matida, Y., and N. Kawasaki, "Study on the Toxicity of Agricultural Control Chemicals in Relation to Freshwater Fisheries Management," No. 2 Toxicity of Agricultural Insecticides to *Daphnia carinata*, King, *Bull. Freshwater Fish Res. Lab.*, Tokyo 8:1-6 (1958). (In: Li and Fleck, 1972.)

Table 23. LC_{50} VALUES (ppb) OF THREE ORGANOPHOSPHATE INSECTICIDES TO BENTHIC INVERTEBRATES

Species	Temperature (°C)	Time (hr) (* = days)	Malathion	Parathion	Methyl parathion	Reference
Stoneflies						
Pteronarcys californica	15.5	24	35	28		a/
	15.5	48	20	11		a/
	21.0	48	21			b/
	48-50°F	48		11		c/
	11-12	48	180	75		d/
	11-12	72	72.5	32.0		d/
	11-12	96	50.0	32.0		d/, e/
	15.5	96	10	5.4		a/
	12.8	5*		36.0		f/
	12.8	10*		2.7		f/
	12.8	15*	45.0	2.2		f/
	12.8	20*	24.0	2.2		f/
	12.8	25*	15.5	2.2		f/
	12.8	30*	8.8	2.2		f/
Acroneuria pacifica	11-12	48	12	6		d/
	11-12	72	16.0	2.9		d/
	11-12	96	7.0	2.8		d/, e/
	12.8	5*	7.7	0.93		f/
	12.8	10*	5.1	0.46		f/
	12.8	15*	3.3	0.45		f/
	12.8	20*	3.2	0.44		f/
	12.8	25*	2.4	0.44		f/
	12.8	30*	0.78	0.44		f/
Pteronarcella badia	15.5	24	10	8.0		a/
	15.5	48	60	5.6		a/
	48-50°F	48	6			b/
	15.5	96	1.1	4.2		a/
Claassenia sabulosa	15.5	24	13	8.8		a/
	15.5	48	6.0	3.5		a/
	15.5	96	2.8	1.5		a/
Caddisflies						
Arctopsyche grandis	51-54°F	96	32	7		e/
Hydropsyche californica	51-54°F	96	22.5	0.43		e/

Table 23. (Continued)

Species	Temperature (°C)	Time (hr) (* = days)	Malathion	Parathion	Methyl parathion	Reference
Mayflies						
Ephemerella grandis	48-50°F	96	100	3		e/
Baetis sp.	21	48	6			b/
Amphipods						
Gammarus lacustris	70°F	24	3.8	12		g/
	70°F	48	1.8	6.0		g/
	60°F	48	1.8	6		c/
	70°F	48	1.0	3.5		g/
	59°F	96	1.62	12.8		e/
Freshwater shrimp	21-27	24		6.6	2.5	h/
Palaemonetes radiakensis	21-27	24		11.8	23.3	h/
Crayfish						
Procambarus clarki	16-32	24			50	i/
	16-32	48			40	i/
	16-32	72			40	i/

Source: Li and Fleck, op. cit. (1972).

a/ Sanders, H. O. and O. B. Cope, "The Relative Toxicities of Several Pesticides to Naiads of Three Species of Stoneflies," *Limnology and Oceanography*, 13(1):112-117 (1968)

b/ Cope, O. B., *op. cit.*, (1966).

c/ FWPCA, *op. cit.*, (1968).

d/ Jensen, L. D. and A. R. Gaufin, "Effects of Ten Organic Insecticides on Two Species of Stonefly Naiads," *Trans. Am. Fish Soc.*, 93:27-34 (1964a).

e/ Gaufin, A. R., L. D. Jensen A. V. Nebeker, T. Nelson, and R. W. Teel, "The Toxicity of Ten Organic Insecticides to Various Aquatic Invertebrates," *Water Sewage Works*, 12:276-79 (1965). (In: Li and Fleck, 1972.)

f/ Jensen, L. D. and A. R. Gaufin, "Long-Term Effects of Organic Insecticides on Two Species of Stonefly Naiads," *Trans. Am. Fish Soc.*, 93:357-63 (1964b). (In: Li and Fleck, 1972.)

g/ Sanders, H. O., "Toxicity of Pesticides to the Crustacean *Gammarus lacustris*," *Technical Papers No. 25* pp. 18, United States Department of the Interior Fish and Wildlife Service (1969).

h/ Nagvi, S. M., and D. E. Ferguson, "Levels of Insecticide Resistance in Freshwater Shrimp, *Palaemonetes kadiakensis*," *Trans. Am. Fish Soc.*, 99:696-99 (1970). (In: Li and Fleck, 1972.)

i/ Muncy, R. J., and A. D. Oliver, Jr., "Toxicity of Ten Insecticides to the Red Crawfish, *Procambarus clarki* (Girard)," *Trans. Am. Fish Soc.*, 92:428-31 (1963). (In: Li and Fleck, 1972.)

The observations by Nicholson et al. (1962) and Grzenda et al. (1962) on the effects of parathion contamination in a farm pond draining a 40-acre watershed grown to peaches treated with parathion have been described in greater detail elsewhere in this subsection (p.139). Based on their careful, comprehensive studies under field conditions representative of a real-life situation, these authors reported that "the fish, zooplankton, aquatic insect, and Oligochaeta populations appeared to be unaffected by parathion residues in pond water and mud. However, there was a significant reduction in immature aquatic insect numbers associated with insecticide use. The data indicate this was caused indirectly by mortality in the adult populations resulting from exposure to parathion residues in the watershed. Zooplankton were not utilized as food by bluegills when immature chironomids were relatively abundant. However, when the insect standing crop became severely reduced, the fish fed almost entirely on planktonic Crustacea."

The studies by Mulla et al. (1963) also described in greater detail above (see p.110) included observation on the effects of parathion on tadpoles of three species of amphibia, i.e., the bullfrog (Rana catesbeiana), the Western toad (Bufo boreas), and Hammond's spadefoot toad (Scaphiopus hammondi). Parathion applied to 1/16-acre field ponds at rates of 0.1 and 0.4 lb AI/acre did not produce mortality when the tadpoles were exposed to the contaminated water for periods of 24 hr.

A number of further studies, in addition to those summarized in Tables 22 and 23, deal with interactions between parathion and aquatic organisms in isolated systems under laboratory conditions.

Zuckerman et al. (1970)[1] found that the alga Chlorella pyrenoidosa proteose degraded parathion in axenic cultures incubated for 7 days at 20°C.

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[1] Zuckerman, B. M., K. Deubert, M. Mackiewicz, and H. Gunner, "Studies on the Biodegradation of Parathion," Plant Soil 33(2):273-81 (1970).

Moore (1970)[1] and Poorman (1973)[2] investigated the effects of parathion on growth and survival of *Euglena gracilis*, a species which they describe as a "photosynthetic microorganism," along with Kortus et al. (1971)[3] ("aquatic alga"), while Gregory et al. (1969)[4] consider *E. gracilis* to be a flagellate.

In Moore's tests, parathion inhibited the growth rate of *E. gracilis* only at the highest rate tested, 1.2 ppm. In the presence of 0.01 M glucose, 10 ppm of parathion did not affect the respiration rate of *E. gracilis* over a period of 80 min.

Poorman found that parathion at 50 and 100 ppm depressed the growth rate of *E. gracilis* only to a small extent during a 24-hr exposure. When the organism was exposed to parathion for 7 days, there was considerable growth stimulation as compared to untreated controls. These results indicate that parathion is not likely to adversely affect *E. gracilis* under field conditions.

Gregory et al. (1969) found that *Euglena gracilis* ("a flagellate"), two species of algae, and two species of ciliates, maintained on culture media at 26°C in an environmental chamber under continuous illumination, concentrated parathion 50 to 116 times when exposed to 1 ppm w/v of the insecticide for 7 days. The supernatant liquids in the cultures contained only small amounts of parathion. The authors attribute this to three possible reasons, i.e., adsorption by the living organism, sorption by nonliving materials, and/or codistillation with water.

Kortus et al. (1971) studied the uptake of ^{32}P-labeled parathion by *Euglena gracilis* maintained under light at 28°C. Parathion uptake was determined at frequent intervals between 1 and 180 min. The maximum uptake of parathion (69%) occurred immediately after addition of the pesticide. The activity decreased precipitously during the following 15 min and continued to drop at a slower pace after that time.

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[1] Moore, R. B., "Effects of Pesticides on Growth and Survival of *Euglena gracilis* Z.," *Bull. Environ. Contam. Toxicol.*, 5(3):226-230 (1970).

[2] Poorman, A. E., "Effects of Pesticides on *Euglena gracilis* I. Growth Studies," *Bull. Environ. Contam. Toxicol.*, 10(1):25-28 (1973).

[3] Kortus, J., P. Macuch, J. Mayer, K. Durcek, and V. Krcmery, "Uptake of 32-P-Parathion and 32-P Imidan," *J. Hyg. Epidemiol. Microbiol. Immunol.*, (Prague) 15(1):101-103 (1971).

[4] Gregory, W. W., Jr., J. K. Reed, and L. E. Priester, Jr., "Accumulation of Parathion and DDT by Some Algae and Protozoa," *J. Protozool.*, 16(1):69-71 (1969).

The data reviewed in this section indicate that parathion is extremely toxic to aquatic insects, highly toxic to the lower aquatic fauna, and relatively nontoxic to the lower aquatic flora. A number of aquatic microorganisms degrade parathion. Many of these organisms also preferentially sorb and thus accumulate parathion from aqueous media. This sorption process may be independent of life processes; sorption rates of live and dead organisms showed little differences.

Effects on Wildlife

Laboratory Studies - Most birds on which controlled experimentation has been done are those which are either important as game animals or are considered nuisances. (Keith and Mulla, 1966;[1] Tucker and Haegele, 1971;[2] Schafer et al., 1973;[3] Rudd and Genelly, 1956;[4] Heath et al., 1972[5]). The use of pesticides in the field noticeably affects songbirds.

The results of the controlled experiments appear to indicate that while the LD_{50} values for toxicity of parathion range from 1 mg/kg to about 24 mg/kg depending on species, with two exceptions, the LD_{50} values were in the range of 1 mg/kg to 5.95 mg/kg (Table 24). The common and scientific names of the avian species are shown in Table 25. With game birds the results recorded in the table indicate that when the species (mallard, pheasant, partridge (Chukar), quail (Japanese)) are ranked from most sensitive to least sensitive, the mallard duck is most sensitive (LD_{50} 1 to 2.13 mg/kg) to parathion, and the Chukar partridge is least sensitive (LD_{50} 24.0 mg/kg) to parathion intoxication.

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[1] Keith, J. O., and M. S. Mulla, "Relative toxicity of Five Organophosphorous Mosquito Larvicides to Ducks," J. Wildlife Manag., 30(3):553-563 (1966).

[2] Tucker, R. K., and M. A. Haegele, "Comparative Acute Oral Toxicity of Pesticides to Six Species of Birds," Toxicol. Appl. Pharmacol., 20:57-65 (1971).

[3] Schafer, E. W., R. B. Brunton, N. F. Lockyer, and J. W. DeGrazio, "Comparative Toxicity of 17 Pesticides to the Quelea, House Sparrow, and Red-Winged Blackbird," Toxicol. Appl. Pharmacol., 26:154-157 (1973).

[4] Rudd, R. L., and R. E. Genelly, Pesticides: Their Use and Toxicity in Relation to Wildlife, State of California Department of Fish and Game, Game Bull. No. 7, pp. 108-113 (1956).

[5] Heath, R. G., J. W. Spann, E. F. Hill, and J. F. Kreitzer, "Comparative Dietary Toxicities of Pesticides to Birds," Bureau of Sport Fisheries and Wildlife, Special Scientific Report, Wildlife No. 152 (February 1972).

Table 24. ACUTE TOXICITY OF PARATHION TO AVIAN SPECIES

	LD_{50} (mg/kg)		
	Oral	Dermal	Reference
Mallard ducks	1.9, 2.13	--	a/, b/
Pheasant	12.4	--	b/
Chukar partridge	24.0	--	b/
Japanese quail	5.95	--	b/
Pigeon	2.52	--	b/
House sparrow	1.3, 3.36	1.8	c/, b/
Quelea	1.8	1.8	c/
Red-winged blackbird	2.4	--	d/, c/
Starling	5.6	--	d/

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a/ Keith and Mulla, *op. cit.* (1966).

b/ Tucker and Haegele, *op. cit.* (1971).

c/ Schafer et al., *op. cit.* (1973).

d/ Schafer, E. W., "The Acute Oral Toxicity of 369 Pesticidal, Pharmaceutical and Other Chemicals to Wild Birds," *Toxicol. Appl. Pharmacol.*, 21:315-330 (1972).

Table 25. COMMON AND SCIENTIFIC NAMES OF BIRDS USED IN CONTROLLED TOXICITY TESTS WITH PARATHION

Common name	Scientific name
Mallard ducks	*Anas platyrhynchos*
Bobwhite quail	*Colinus virginianus*
Japanese quail	*Coturnix coturnix japonica*
Ring-necked pheasant	*Phasianus colchicus*
Chukar	*Alectoris graeca*
Pigeon	*Columba livia*
House sparrow	*Passer domesticus*
Red-winged blackbird	*Agelaius phoeniceus*
Quelea	*Quelea quelea*
Pintail ducks	*Anas acuta*
Starling	*Sturnus vulgaris*

The information on dermal toxicity for wild birds is limited; only one paper was found (Schafer et al., 1973). These investigators studied the oral and dermal toxicity of 17 chemicals on Quelea, the house sparrow, and on the red-winged blackbird. The acute oral and dermal LD_{50} values for Quelea exposed to parathion were 1.8 mg/kg and 1.8 mg/kg, respectively. The acute oral and dermal LD_{50} values for the house sparrow were 1.3 mg/kg and 1.8 mg/kg, respectively.

While oral toxicity values are important and laboratory measurements of lethality are basic in predicting the immediate impact of a pesticide on the environment, measurements of dermal toxicity would appear to be equally if not more important, in that wildlife mortality has been reported by some to occur chiefly through contact with foliage (Rudd and Genelly, 1956). However, based on the limited amount of data shown in Table 24, there is little difference between the oral and dermal LD_{50} values.

The toxic effect of feeding parathion to four species of birds was studied by Heath et al. (1972). These birds were at least 9 days old before administration of the diets carrying the chemical. Data from this study are summarized in Table 26.

Table 26. AVIAN DIETARY TOXICITY (8 Days)

	LC_{50} in feed (ppm) (95% conf. limits)	
	Methyl Parathion	Parathion
Bobwhite quail	90 (73-111)	194 (150-245)
Japanese quail	46 (38-55)	44 (36-53)
Pheasant	116 (101-134)	365 (316-420)
Mallard	682 (541-892)	275 (183-373)

Data of Heath et al., op. cit. (1972).

The Japanese quail were more susceptible than bobwhite quail, pheasant or mallard ducks to parathion poisoning. It is interesting to note that it required nearly five times as much parathion to kill the bobwhite species as it did to kill the Japanese quail.

One of the difficulties in fully assessing the hazard potential for wild birds from laboratory studies is emphasized by the statement of Heath et al. (1972): "our particular interest, however, was in dietary toxicity because ingestion is undoubtedly the predominant route of exposure in wild species." This statement is in opposition to that of Rudd and Genelly (1956): "Wildlife mortality occurs chiefly through contact with foliage." Regardless of the most probable route of entry for parathion in wild birds, the age of the birds at the time of exposure has a decided influence on the outcome of the exposure.

Ducks from 36 hr of age to 6 months of age have been used to determine the relative toxicity of parathion for birds in these different age groups. The results of this study by Hudson et al. (1972)[1/] are shown in Table 27.

Table 27. EFFECT OF AGE ON PARATHION TOXICITY IN MALLARD DUCKS[a/]

Approximate age of ducks	LD_{50} (mg/kg)
36 hr	1.65
7 days	1.44
30 days	1.65
6 months	2.34

[a/] Data from Hudson et al., op. cit. (1972).

The lethality does not appear to change between 36-hr ducklings (LD_{50} 1.65 mg/kg) and 1-month-old birds. However, as the ducks mature, they become less susceptible to parathion intoxication (LD_{50} 2.34 mg/kg--6 months). This is in agreement with the findings of Brodeur and DuBois (1963),[2/] who concluded that young rats were almost always more susceptible to anticholinesterase insecticides than older animals.

[1/] Hudson, R. H., R. K. Tucker, and M. A. Haegele, "Effect of Age on Sensitivity: Acute Oral Toxicity of 14 Pesticides to Mallard Ducks of Several Ages," Toxicol. Appl. Pharmacol., 22:556-561 (1972).

[2/] Brodeur, J., and K. P. DuBois, "Comparison of Acute Toxicity of Anticholinesterase Insecticides to Weanling and Adult Male Rats," Proc. Soc. Exp. Biol. Med., 114(2):509-511 (November 1963).

Although bradycardia is generally reported in parathion poisoning in mammals, tachycardia is found to be a more consistent response to parathion poisoning with avian species such as quail and ducks. After tachycardia commenced, the respiratory rate increased and remained high until death occurred. In both mallards and quail a small but consistent rise occurs in the hematocrit (see Table 28). It was observed that parathion poisoning did not significantly alter the blood pressure in the two avian species (McFarland and Lacy, 1968).[1]

Table 28. EFFECTS OF INTRAVENOUS INJECTIONS OF PARATHION ON HEMATOCRIT OF DUCKS AND QUAIL[a]

Hematocrit	Mallard ducks (36)[b]		Japanese quail (46)	
	Males	Females	Males	Females
Before parathion exposure	48.2	47.3	47.6	45.1
After parathion exposure	51.8	50.5	50.8	47.6

[a] Data from McFarland and Lacy, op. cit. (1968).

[b] Number of birds.

The birds mentioned in Table were given intravenous injections as follows: mallards, 0.61 to 1.70 mg/kg; quail 3.9 to 5.7 mg/kg.

Field Studies - Robel et al. (1972)[2] reported on the effects of parathion and other insecticides on populations of wild rodents in Kansas, based on observations made from 1965 to 1969. The study was conducted on two sites in Ellis County, Kansas, in a newly created irrigation district that had not been intensively cultivated prior to 1965. No insecticidal residues were found in samples of water, soil, plants, or animals collected from the study sites prior to initiation of the study in 1965. Parathion was applied to one field (the second field served as an untreated control) at recommended and commonly used rates of application of 0.38, 1.4, 0.79, and 1.01 lb AI/acre in 1965, 1966, 1967, and 1968, respectively. Live rodents were trapped in the treated and control field from 1965 through 1969. A total of 4,661 rodents were captured, of which 162 were analyzed for residues.

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[1] McFarland, L. Z., and P. B. Lacy, "Acute Anticholinesterase Toxicity in Ducks and Japanese Quail," Toxicol. Appl. Pharmacol., 12:105-114 (1968).

[2] Robel, R. J., C. D. Stalling, M. E. Westfahl, and A. M. Kadoum, "Effects of Insecticides on Populations of Rodents in Kansas - 1965-69," Pest. Monit. J., 6(2):115-121 (September 1972).

No parathion residues were found in any of these specimens. The species composition of the trapped rodents was similar for the treated and the untreated study area, as were the population levels of Peromyscus maniculatus, which comprised about 74% of the total rodent population in the two areas. Average minimal longevity for P. maniculatus and monthly survival between June and September did not differ significantly between the treated and untreated area. Thus, none of the parameters observed in this study indicated any effects on the wild rodent population from the use of parathion at field rates over a 4-year period.

Bejer-Petersen et al. (1972)[1/] studied the effects of spray treatments of parathion and other insecticides in forests on birds living in nest boxes. Parathion spraying (rate not given in abstract) was carried out in 1965 and in 1967 at a time when nestlings of Parus major and P. ater were most numerous. The loss rate of Parus nestlings was adversely affected by the parathion treatment. The breeding success of Ficedula hypoleuca, the third species in these tests, was 50%, compared to 64 to 100% in control areas. Parathion residues in these birds ranged from traces to 11 ppm. In several of the broods of each species, reduced brain cholinesterase activity was observed. The sprayings had no effect on bird populations in the years following the treatment.

Bucknell (1970)[2/] reported deaths of a number of species of birds following the application of parathion in granulated form. The bird species most frequently affected were magpies, gulls, hawks, blackbirds, thrushes, and finches.

The summary on parathion by Pimentel (1971)[3/] includes the following three reports on wildlife toxicity.

[1/] Bejer-Petersen, B., R. R. Hermansen, and M. Weihe, "On the Effects of Insecticide Sprayings in Forests on Birds Living in Nest Boxes," Dan. Ornithol. Foren. Iidsskr., 66(1,2):30-50 (1972).

[2/] Bucknell, E. S., "Side-Effects of Granulated Insecticides in Canterbury," Proc. N. Z. Weed Pest Contr. Conf. No. 23, pp. 124-126 (1970).

[3/] Pimentel, D., "Ecological Effects of Pesticides on Nontarget Species," Executive Office of the President, Office of Science and Technology, U.S. Government Printing Office, Washington, D.C. (1971).

Buttiker (1961)[1] reported on a large-scale spray program carried out on a citrus estate in the Union of South Africa. Parathion was applied at 7.5 lb AI/acre for the control of citrus insects. In the treated grove, nearly 800 birds were found dead.

Mulla (1966)[2] reported that mallard ducks appeared to be unaffected when duck ponds were treated with parathion at the rate of 0.85 lb AI/acre.

In a USDI (1966)[3] study, parathion was applied at rates of 0.5 and 3.0 lb AI/acre. Pheasants were not affected by the lower rate, but there was about 10% mortality at the 3.0-lb rate.

Our search of the literature and of other information sources failed to yield additional data on the toxicity of parathion to wildlife under field conditions. The data on the oral acute toxicity of parathion to wildlife summarized by Tucker and Crabtree (1970)[4] indicate that parathion is highly toxic to all species studied. This is also evident from the data on the toxicity of parathion to wildlife summarized by Pimentel (1971).

Parathion labels carry the warning "Poisonous to fish and wildlife. This product is toxic to fish and wildlife. Birds and other wildlife in treated areas may be killed."

Effects on Beneficial Insects

Bees - Several investigators have reported on the toxicity of parathion to bees (*Apis mellifera*) by direct topical application, or by exposing bees to sugar solutions containing parathion, to parathion deposits on inert surfaces, or to vapors from parathion residues. By all of these routes of application, parathion is highly toxic to bees.

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[1] Buttiker, W., "Ecological Effects of Insect Control on Bird Populations, "Int. Union Conserva. Nature Natur. Resources, Tech. Meeting, Warsaw 1960, *Proc.*, 8:48-60 (1961). Quoted from: Pimenta Pimentel (1971).

[2] Mulla, M. S., "Vector Control Technology and Its Relationship to the Environment and Wildlife," *J. Appl. Ecol.*, 3(Supplement on Pesticides in the Environment and Their Effects on Wildlife):21-28 (1966). Quoted from: Pimentel (1971).

[3] USDI, "Wildlife Research; Problems, Programs and Progress. Pesticide-Wildlife Relations," Fish and Wildlife Serv., Bureau Sport Fish Wildlife Circ. 43, 117 pp. (1966). Quoted from: Pimentel (1971).

[4] Tucker, R. K., and D. G. Crabtree, "Handbook of Toxicity of Pesticide to Wildlife," U.S. Department of the Interior, Fish and Wildlife Service, Denver Wildlife Research Center, Resource Publication No. 84, p. 82 (1970).

The *Handbook of Toxicology* (WADC, 1959)[1] states: "Parathion is perhaps the most generally potent insecticide presently in commerce." This is clearly supported by tabular listings of the effects of lethal dosages of parathion on a considerable number of insects.

Stephen (1972)[2] recently studied the effects of chronic sublethal doses of parathion on the behavior of the leafcutter bee (*Megachile rotundata*). Bees emerging from diapause were treated topically with sublethal doses of parathion (1 µg/g) at 2-or 3-hr intervals over a 24-hr period. The authors concluded that the rhythmic responses to parathion appeared to be light-cued, while the response to dieldrin, another pesticide tested in the same manner, was temperature-dependent. In a companion study on the effect of parathion on the ability of the leafcutter bee to synchronize its activity rhythm to the environment, there was no evidence of the clock being affected. Web spinning behavior of the spider, *Araneus diadamatus*, was not affected by a single application of parathion, but the amount of silk available was reduced.

Barker (1970)[3] investigated whether the classical antidotes against parathion poisoning in mammals, atropine sulfate and N-methylpyridinium-2-aldoxime (PAM), would work on honeybees. When parathion was applied to the thorax of bees, the 24-hr LD_{50} was 0.08 to 0.16 µg/bee. Treated bees showed no reduction in mortality when atropine sulfate with or without salts of PAM was fed, applied topically, or injected. Thus, these parathion antidotes were ineffective on bees, at least under the conditions of this experiment.

Johansen (1972a,b)[4,5] investigated the toxicity of field-weathered residues of parathion and other insecticides to different species of bees.

1/ WADC, "Insecticides," *The Handbook of Toxicology*, Vol. III, Wright-Patterson Air Force Base, Ohio Air Force Systems Command, WDAC Tech. Report 55-16 (1959).

2/ Stephen, W. P., "The Effects of Chronic Sublethal Doses of Pesticides on Behavior and Longevity in Arthropods," *Oregon State Univ. Environ. Health Sci. Cent. Annu. Progr. Rep.*, pp. 161-167 (1972).

3/ Barker, R. J., "Cholinesterase Reactivators Tested as Antidotes for Use on Poisoned Honeybees," *J. Econ. Entomol.*, 63(6):1831-1833 (December 1970).

4/ Johansen, C. A., "Spray Additives for Insecticidal Selectivity to Injurious vs Beneficial Insects," *Environ. Entomol.*, 1(1):51-54 (February 1972a).

5/ Johansen, C. A., "Toxicity of Field-Weathered Insecticide Residues to Four Kinds of Bees," *Environ. Entomol.*, 1(3):393-394 (June 1972b).

Parathion from a 4-lb/gal emulsifiable liquid was applied to alfalfa at the rate of 0.5 lb AI/acre. Three kinds of bees were exposed to the parathion residues 10 hr after application. Bee mortality was determined after 24 hr and ranged from 41 to 66%. When bees were exposed to 3-hr old residues of parathion on alfalfa treated at 0.5 lb AI/acre, mortalities ranged from 91 to 100%. On 8-hr old residues, mortalities ranged from 61 to 93%.

The entomological literature contains many other records of the toxicity of parathion to bees. Within the limits of time and resources available for this review, it was not possible, and was not considered of prime importance to collect and review the entire literature on the toxicity of parathion to bees. The high toxicity of this insecticide to bees has been well established and is universally recognized. Parathion labels state: "This product is highly toxic to bees exposed to direct treatment or residues on crops." Many extension service recommendations concerning parathion carry specific warnings applicable to local conditions, crops, etc.

Parasites and Predators - The importance of naturally occurring parasites and predators of insect and mite pests in suppressing these pests and reducing or preventing economic damage from their activities has been increasingly recognized in recent years. A number of investigators have studied the effects of parathion on such parasites and predators, mostly under laboratory conditions.

Hamilton and Kieckhefer (1969)[1] investigated the toxicity of parathion to predators of the English grain aphid (Macrosiphum avenae). Adult and larval forms of the three most numerous and ubiquitous predators of cereal aphids in South Dakota, Hippodamia convergens (the convergent lady beetle), Nabis americoferus, and Chrysopa carnea were field-collected for laboratory mortality tests. By topical application to adults, the LD_{50} of parathion to the aphid was 1.8 µg/g, while it was 21 µg/g to N. americoferus and 40 µg/g to H. convergens. LC_{50} values of parathion to the same insects were determined by exposing the insects to 4 and 24-hr-old deposits of appropriate concentrations. Again, the LC_{50} values of parathion to the predators were much higher than to the aphid.

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[1] Hamilton, E. W., and R. W. Kieckhefer, "Toxicity of Malathion and Parathion to Predators of the English Grain Aphid," J. Econ. Entomol., 62(5):1190-1192 (October 1969).

Croft and Jeppson (1970)[1] studied the toxicity of parathion and other insecticides to *Typhlodromus occidentalis*, an important natural enemy of plant feeding mites in the Western United States. Strains of this phytoseiid mite from Washington, Utah, and California were studied at the University of California insectary. All predator strains were uniformly tolerant to field use rates of parathion. The results clearly indicate that strains of this predator from four different locations have developed a high degree of tolerance to parathion.

Moffitt et al. (1972)[2] studied the toxicity of parathion and several other frequently used orchard pesticides to the convergent lady beetle (*Hippodamia convergens*), one of the more common and important predators of aphids in deciduous fruit orchards in Washington. When adult beetles were sprayed directly with parathion at 0.125 and 0.25 lb AI/100 gal. or exposed to residues from parathion sprays of these concentrations, none survived. In orchard tests, percent survival of *H. convergens* adults exposed to residues of parathion applied at 0.125 and 0.25 lb AI/100 gal.was zero when the beetles were exposed for 2 days to 2-day-old residues. When exposed for 2 days to residues 9 days old, 83% of the beetles survived in one test, none in another. Only 2% survived exposure to 12-day-old deposits for 5 days.

The LD_{50} of parathion to adult beetles (topical application) was 0.062 µg/beetle.

These data indicate that parathion applications at the above rates would not be compatible with survival of adult beetles under field conditions.

Lingappa et al. (1972)[3] studied the effects of parathion on three developmental stages of *Lysiphlebus testaceipes*, a parasite of the greenbug (*Schizaphis graminum*). Field-collected parasites were used to experimentally parasitize greenbugs. One, four, and eight days after parasitization, the greenbugs were transferred to sorghum plants about

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[1] Croft, B. A., and L. R. Jeppson, "Comparative Studies on Four Strains of *Typhlodromus occidentalis*. II. Laboratory Toxicity of Ten Compounds Common to Apple Pest Control," *J. Econ. Entomol.*, 63(5):1528-1531 (October 1970).

[2] Moffitt, H. R., E. W. Anthon, and L. O. Smith, "Toxicity of Several Commonly Used Orchard Pesticides to Adult *Hippodamia convergens*," *Environ. Entomol.*, 1(1):20-23 (February 1972).

[3] Lingappa, S. S., K. J. Starks, and R. D. Eikenbary, "Insecticidal Effect on *Lysiphlebus testaceipes*, a Parasite of the Greenbug, at Three Development Stages," *Environ. Entomol.*, 1(4):520-521 (August 1972).

10 days old and sprayed in the laboratory with parathion at the equivalent of 0.25 lb AI/acre, the common greenbug control dosage. Adult parasites failed to emerge from the greenbugs that were exposed to parathion 1 and 4 days after parasitism, while untreated controls had 94.1 and 93.9% emergence, respectively. Parasite emergence from the greenbugs exposed to parathion 8 days after parasitization was 76.5% (97.8% in the untreated control). The authors suggest that apparently the host greenbugs were killed by the parathion treatment before the parasite had completed larval development or, in the case of the 1-day-old treatments, even before parasite eggs had hatched. Thus, field sprays of parathion at these early states in the life cycle of the parasite would be distinctly detrimental to parasite survival.

Several reports from overseas also deal with the effects of parathion on beneficial insects. Gupta and Kushwaha (1970)[1/] found that parathion and several other insecticides were toxic to the aphid predator Menochilus sexmaculata. Satpathy et al. (1968)[2/] studied the toxicity of parathion and seven other insecticides to the aphid predator Chilomenes sexmaculata by feeding adult beetles with insecticide poisoned aphids. Parathion was among the insecticides most toxic to the predator. Teotia and Tiwari (1972)[3/] also found parathion to be among the insecticides most toxic to the aphid predator Coccinella septembpunctata.

The reports summarized in this section indicate that parathion would permit survival of beneficial insects in some crop pest predator/parasite systems, but certainly not in all. The relative toxicity of the insecticide to the pest and to the predators and parasites and their respective life stages will have to be studied on a case-by-case basis. At least some predators and parasites appear to be capable of developing tolerance or resistance to parathion in the same manner as many insect and mite pests.

[1/] Gupta, R. S., and K. S. Kushwaha, "Toxicity of Some Insecticides to the Predator, Menochilus sexmaculata," Indian J. Entomol., 32(Pt. 4):379-381 (1970).

[2/] Satpathy, J. M., G. K. Padhi, and D. N. Dutta, "Toxicity of Eight Insecticides to the Coccinellid Predator Chilomenes sexmaculata," Indian J. Entomol., 30(1):130-132 (1968).

[3/] Teotia, T. P. S., and G. C. Tiwari, "Toxicity of Some Important Insecticides to the Coccinellid Predator, Coccinella septempunctata," Labdev, Part B, 10(1):17-18 (1972).

Interactions with Lower Terrestrial Organisms

Reviews - The relationships between insecticides and microorganisms have recently been reviewed by several authors. Matsumura and Boush (1971)[1] point out that organic phosphate pesticides (including parathion) have thus far apparently neither presented serious problems in soils as regards undesirable persistence, nor demonstrated an extraordinary affinity for fat with resulting concentration within a food chain. Although considerable variations exist between individual organophosphates, most of them are readily degraded in soil, mainly by hydrolytic and oxidative means.

These authors also point out that, although several workers have demonstrated in the laboratory that some microorganisms are able to degrade even the most stable and persistent organic insecticides, no one has yet demonstrated whether or not this occurs in nature, or even that these compounds serve as nutritional or energy sources for organisms. "In fact there are no reports as yet that these chemicals have been shown to serve as sole nutritional carbon sources." The incredible biological and chemical complexity of the soil is a formidable obstacle to gaining an understanding of the fate of pesticides in that habitat, and of the mechanisms involved in their metabolism and degradation *in situ*.

Another recent review of the interactions between pesticides (including parathion) and the soil fauna was authored by Drift (1970).[2]

Ecology, degradation and movement of pesticides in the soil were reviewed in an international symposium at Michigan State University in 1970. The proceedings of this symposium have been published and contain a wealth of general data and overviews of different aspects of the subject, but no reports dealing specifically with parathion.

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[1] Matsumura, F., and G. M. Boush, "Metabolism of Insecticides by Microorganisms," *Soil Biochem.*, Vol. II pp. 320-336, Marcel Decker, New York (1971).

[2] Drift, J., "Pesticides and Soil Fauna," *Meded. Rijksfac. Landbouwwetensch.*, Gent, 35(2):707-716 (1970).

Field Studies - Bollen et al. (1954)[1] investigated the effects of field treatments of insecticides, including parathion, on the numbers of bacteria, Streptomyces, and molds in field plots in Oregon. Parathion in the form of a wettable powder was incorporated with the soil by rotary tillage at the rate of 10 lb AI/acre. Composite soil samples at plow depth were taken on the day of application and transported to the laboratory in paper bags or friction-top tin cans. These containers were opened and allowed to stand, well isolated from each other, until sufficiently dry for screening. The samples were then passed through a 10-mesh sieve and stored in friction-top cans at room temperature for 10 to 20 days, when they were counted. The counts of molds and bacteria in the parathion-treated samples differed very little from those in the untreated controls.

Hubbell et al. (1973)[2] studied the effects of several pesticides, including parathion, on the relative numbers of microbes and on nitrification in field plots to which the pesticides, alone and in combination, had been applied at times and rates of application approximating current agronomic practice in the growing of shadeleaf tobacco in the area of Quincy, Florida. Numbers of microbes and nitrification were monitored at 2-week intervals for 16 weeks following application. There were no significant effects from the pesticide treatments on the relative numbers of bacteria and actinomycetes. The count of fungi increased in the parathion + DDT + Zineb treatment. There were no significant effects on nitrification.

Wolfe et al. (1973)[3] investigated the effects of very high levels of parathion on soil microorganisms. The extreme concentrations of parathion in the soil studied by these authors resulted from simulated spillage of concentrated parathion emulsifiable liquid and dilute sprays. Soil from a test plot that had received a topical application of 45.5% emulsifiable concentrate of parathion 2 years earlier contained 3.4% parathion, a concentration equivalent to 136,000 lb AI/6 acre-ft. Soil from this plot and soil from a comparable, untreated control plot were plated on nutrient yeast-dextrose agar containing different levels of parathion, to determine if the soil organisms might have developed resistance or tolerance to the insecticide. The number of colonies that developed in 6 days at 70°F was used as an indicator. Significantly fewer colonies of microorganisms were isolated from the treated soil. Microorganisms from the treated soil showed no evidence of resistance to parathion.

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[1] Bollen, W. B., H. E. Morrison, and H. H. Crowell, "Effect of Field Treatments of Insecticides on Numbers of Bacteria, Streptomyces, and Molds in the Soil," J. Econ. Entomol., 47(2):302-306 (1954).

[2] Hubbell, D. H., D. F. Rothwell, W. B. Wheeler, W. B. Tappan, and F. M. Rhoads, "Microbiological Effects and Persistence of Some Pesticide Combinations in Soil," J. Environ. Qual., 2(1):96-99 (1973).

[3] Wolfe, H. R., D. C. Staiff, J. F. Armstrong, and S. W. Comer, "Persistence of Parathion in Soil," Bull. Environ. Contam. Toxicol., 10(1):1-9 (1973).

These data indicate that soil organisms survive extremely high concentrations of parathion in the soil, even though they were adversely affected.

Laboratory Studies - Several authors have investigated interactions between parathion and isolated microorganisms under laboratory conditions.

Mick and Dahm (1970)[1/] incubated parathion at concentrations in the range of 10^{-4} to 10^{-6}M with cultures of two species of Rhizobium and found that these fungi metabolized the insecticide primarily by nitroreduction. Eighty-five percent of the initial parathion was reduced to aminoparathion. About 10% was hydrolyzed to the corresponding phosphorothioic acid. Both of these degradation products have negligible toxicity compared to parathion. No paraoxon was detected. Populations of both Rhizobium species decreased somewhat in the presence of parathion in the incubation mixtures.

Mackiewicz et al. (1969)[2/] investigated the biodegradation of parathion in a germ-free system. Bean roots grown under aseptic conditions were exposed to parathion for 7 days with or without the alga Chlorella. The only pesticide-related product found in extracts from the aerial parts of plants exposed to the insecticide alone was parathion.

[1/] Mick, D. L., and P. A. Dahm, "Metabolism of Parathion by Two Species of Rhizobium," J. Econ. Entomol., 63(4):1155-1159 (August 1970).

[2/] Mackiewicz, M., K. H. Deubert, H. B. Gunner, and B. M. Zuckerman, "Study of Parathion Biodegradation Using Gnotobiotic Techniques," J. Agr. Food Chem., 17(1):129-130 (January/February 1969).

By contrast, parathion and a sulfur-containing parathion metabolite were recovered from bean leaves and stems of plants whose roots were exposed to both parathion and the algae. The authors conclude that parathion metabolites found in plants may be a product of microbial metabolism, rather than metabolism by the plant as has generally been assumed by other authors. They emphasize the utility of, and often the requirement for, germ-free systems in the study of the degradative pathways of pesticides.

Cowley and Lichtenstein (1970)[1] studied the toxic effects of parathion and other insecticides on 17 species of fungi from Wisconsin prairie soils. The fungi, including Aspergillus fumigatus and Fusarium oxysorum, were grown on nutrient agar that had been treated with the insecticides at six rates, ranging from 1 to 40 ug/ml. The pesticide-containing inoculated culture media were incubated for 5 days at about 34°C. The dry weight of each colony was then determined as a measure for the toxicity of the insecticides. In further, similar tests, yeast extract, vitamins, and nitrogenous compounds were added to the cultures to study possible counteractions of insecticidal effects. Relative sporulation was evaluated by visual inspections, and the diameter of each fungal colony was also recorded.

Parathion and all other insecticides inhibited the growth of most of the fungal species. Threshold concentrations at which no decrease in fungal growth occurred differed for each compound, and also for each of the two fungal species. None of the fungi tested were able to use parathion or any of the other insecticides as a source of carbon or phosphorus.

Anderson (1971)[2] investigated the capacity of several fungi isolated from an agricultural loam soil to degrade DDT or dieldrin. He reported that Mucor alternans in shake cultures partially degraded DDT within 2 to 4 days into two water-soluble metabolites. At a concentration of 1 ppm, parathion partially inhibited DDT degradation by this fungus, although fungal growth was not drastically affected. A parathion hydrolysis product, p-nitrophenol, had no effect on fungal growth or DDT degradation.

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[1] Cowley, G. T., and E. P. Lichtenstein, "Growth Inhibition of Soil Fungi by Insecticides and Annulment of Inhibition by Yeast Extract or Nitrogenous Nutrients," J. Gen. Microbiol., 62(1):27-34 (1970).

[2] Anderson, J. P. E., "Factors Influencing Insecticide Degradation by a Soil Fungus, Mucor Alternans," Diss. Abstr. Int., 32(6):3114B-3115B (1971).

Sethunathan and Yoshida (1972, 1973)[1,2] found that parathion and several other organic phosphate insecticides were rapidly hydrolyzed by a cell-free extract of a species of Flavobacterium isolated from water of a rice field that had previously been treated with diazinon. Most of the parathion was rapidly decomposed (within 30 min), without a lag phase. The major metabolite formed was *p*-nitrophenol which was not further degraded by the organism. No appreciable degradation of aminoparathion was observed.

Griffiths and Walker (1970)[3] investigated the microbiological degradation of parathion in soil percolation experiments. Parathion in the soil was degraded by a heat labile agent which was transferable in small amounts between soil cultures. This observation is consistent with the effects of microorganisms, but the responsible organisms have not been isolated in pure cultures.

Residues in Soil

Laboratory Studies - Iwata et al. (1973)[4] investigated the persistence of parathion in six California soils under laboratory conditions. Each soil was fortified with 20 ppm (equivalent to 80 lb AI/6 acre-foot) of parathion, kept in enameled trays, and maintained at 30°C and a soil moisture level of approximately 40% of saturation. In four of the six soils (Mocho silt loam, Linne clay, Madera sandy loam, and Laveen loamy sand), residues decreased rapidly, i.e., from 20 ppm to 0.2-2.0 ppm in 30 days. Chemical hydrolysis and microbial degradation were responsible for the rapid decline. In Windy loam, the residue remained above 3.0 ppm after 8 months. In two experiments differing slightly in soil

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[1] Sethunathan, N., and T. Yoshida, "Conversion of Parathion to Paranitrophenol by Diazinon Degrading Bacterium," Proc. Inst. Environ. Sci. Annu. Tech. Meet. 18:255-257 (1972).

[2] Sethunathan, N., "Degradation of Parathion in Flooded Acid Soils," J. Agr. Food Chem., 21(4):602-604 (1973).

[3] Griffiths, D. C., and N. Walker, "Microbiological Degradation of Parathion," Meded. Rijksfac. Landbouwwetensch., Gent, 35(2):805-810 (1970).

[4] Iwata, Y., W. E. Westlake, and F. A. Gunther, "Persistence of Parathion in Six California Soils Under Laboratory Conditions," Arch. Environ. Contam. Toxicol., 1(1):84-96 (1973).

moisture, the residue in Santa Lucia silt loam was about 1.5 ppm after 8 months in one experiment, and about 0.5 ppm after 6 months in the other. The latter two soil types gave linear semilogarithmic persistence curves, suggestive of degradation by hydrolysis. The degradation of the parathion residues remaining in Windy loam (3.2 ppm) and Santa Lucia silt loam (2.2 ppm) 7.7 months after fortification was not greatly accelerated by flooding with water. Parathion disappeared more rapidly in soils with low organic matter, suggesting that binding to organic matter may reduce the availability of parathion for degradation.

According to the authors, the results of this study support the conclusion of other investigators that most of the parathion applied to soil disappears rapidly, but that low levels may be retained by the soil for extremely long periods. The persistence of parathion is partially dependent on soil types. In soils with strong microbial activity, rapid biological degradation tends to overshadow differences in soil types. In situations where hydrolysis is the primary degradative mechanism, differences in soil type exert greater influence.

Campbell et al. (1971)[1/] studied the influence of organic matter content of soils on the efficacy of parathion against the wireworm (Melanotus communis) in the laboratory. Parathion and most of the other insecticides tested were affected by the organic matter content of the soil; wireworm control increased with decreasing organic matter content. These observations support the results of other studies, including those by Iwata et al. (1973) discussed above, that soil organic matter binds parathion residues, reducing their availability for insect control as well as degradation.

Weidhaas et al. (1961)[2/] reported on the adsorption of parathion by soil from water dispersions and vermiculite granules. Different volumes of water (100 or 250 ml) containing 0.02 ppm of parathion were added to jars containing 50 g of soil that had been premoistened with 25 ml of untreated water. Changes in the insecticide content of the supernatant water and of the soil were monitored by analyzing extracts, 0, 6, and 24 hr after preparation. Immediately after initiation of the

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[1/] Campbell, W. V., D. A. Mount, and B. S. Heming, "Influence of Organic Matter Content of Soils on Insecticidal Control of the Wireworm," J. Econ. Entomol., 64(1):41-44 (1971).

[2/] Weidhaas, D. E., M. C. Bowman, and C. H. Schmidt, "Loss of Parathion and DDT to Soil from Aqueous Dispersions and Vermiculite Granules," J. Econ. Entomol., 54(1):175-177 (1961).

test, most of the parathion was found in the water, but the amount in the soil increased as time elapsed, indicating soil adsorption. When parathion was applied in formulations on vermiculite granules containing 0.01 and 0.025% of AI (weight/weight), it was readily released into water from the granules. The radiometric results were supplemented by bioassay tests of extracts of water and soil with mosquito larvae. The results show that parathion dispersed in water looses insecticidal toxicity in the presence of soil, and that this loss results primarily from adsorption on the soil.

Harris (1966)[1/] investigated the bioactivity of parathion and other insecticides in 10 soil types, employing a bioassay technique using first instar nymphs of the common field cricket (_Acheta pennsylvanicus_) as the test insect. Soils were tested both dry and moist. In most soils, bioactivity was dependent on the organic content of the soil. Parathion was 1,132 times less active in a muck soil containing 64.6% organic matter as compared to quartz sand. There was no direct correlation between organic content and bioactivity in dry soils. The presence of moisture activated parathion in mineral soils, but not in muck soils. The author concluded that in muck soils, parathion (and other insecticides) appear to be sorbed in such a fashion that they are not released from active sites on soil particles in the presence of moisture. Between different pesticides (chlorinated hydrocarbon and organophosphate insecticides) and different soil fractions (clay, sand, silt, and organic), at least three different bonding mechanisms appear to exist.

Burkhardt and Fairchild (1967a)[2/] also employed a cricket species, _Acheta domesticus_, as bioassay test insects in laboratory studies designed to determine the bioactivity of parathion and other insecticides in five soil types, each at two moisture levels. The insecticidal activity of parathion (and of the other insecticides tested) varied with soil type and moisture content. Highest initial insect mortality was obtained in the sandy soils, but the residual activity was shorter in these. Increased moisture generally resulted in increased insect mortality.

[1/] Harris, C. R., "Influence of Soil Type on the Insecticidal Activity of Insecticides in Soil," _J. Econ. Entomol._, 59(5):1221-1225 (1966).

[2/] Burkhardt, C. C., and M. L. Fairchild, "Toxicity of Insecticides to House Crickets and Bioassay of Treated Soils in the Laboratory," _J. Econ. Entomol._, 60(6):1496-1503 (1967a).

Lichtenstein et al. (1966)[1] studied the effects of detergents on the persistence of parathion in the soil. The addition of alkyl benzene sulfonate detergents to insecticide-treated soils increased the persistence of parathion. Parathion was applied to Carrington silt loam free of insecticidal residues at the rate of 10 ppm. The treated soils, with and without detergents added, were incubated for 15 days at 30°C. At that time, 28.5% of the amount of parathion applied was recovered from the soil without added detergents, while 79.5 and 80.0%, respectively, of the applied rate were recovered from soils to which two different detergents had been added. Soils not treated with the detergents had an average count of 4.33 million bacteria per gram of dry soil, while those treated with the detergents contained 56.7 and 228 million, respectively, 15 days after incubation.

In a further experiment, parathion was applied to a loam soil and a quartz sand at the rate of 10 ppm with or without detergents. Two months after the insecticidal and detergent applications, 13 times more parathion was recovered from the loam soil that had been treated with detergent, as compared to the series free of detergents. In the quartz sand, only 0.5 to 3 times more parathion was recovered from the detergent-containing as compared to the detergent-free systems.

The effects of detergents and inorganic salts in water on the persistence and movement of parathion in soils were investigated by Lichtenstein et al. (1967).[2] Quartz sand of low sorptive capacity and minimal microbiological activity served as control. The effects of the sorptive capacities of a loam soil became evident. Water that had been treated at the rate of 2 lb of parathion per 6-in. acre contained only small amounts of parathion after it had been percolated through untreated loam soil. The amount of parathion found in the percolated water was a function of the concentration of the insecticide in the soil. When detergents (alkyl benzene sulfonates) were added to the system, they increased the persistence of parathion in the soil and the amount of parathion in the percolated water. When untreated water, with or without added detergents, was percolated through soil treated with parathion at the rate of 20 lb AI/acre, parathion concentrations in the percolated water samples ranged from 0.2 to 0.8 ppb.

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[1] Lichtenstein, E. P., K. R. Schulz, R. F. Skrentny, and Y. Tsukano, "Toxicity and Fate of Insecticide Residues in Water," Arch. Environ. Health, 12:199-212 (1966).

[2] Lichtenstein, E. P., T. W. Fuhremann, K. R. Schulz, and R. F. Skrentny, "Effect of Detergents and Inorganic Salts in Water on the Persistence and Movement of Insecticides in Soils," J. Econ. Entomol., 60(6):1714-1721 (1967).

These amounts are far below the water solubility of parathion, and the water was not toxic to mosquito larvae. When salts were added to the percolating water, it reduced the amount of parathion residues found in it.

From these results, the authors conclude that it appears unlikely that water in deeper soil strata could become contaminated with insecticidal residues from the upper agricultural soil layers.

The behavior of parathion when applied to the surface of soils was studied in flow-through lysimeters by Herzel (1971).[1] The lysimeters, 1.35 m long, were packed with sand or sandy loam, with or without a 20-cm covering of humus, treated with parathion and the other insecticides at recommended rates, and watered with the equivalent of normal or excessive rainfall. Samples of the runoff and percolated water collected throughout the test, and soil cores taken at the end of the 8-month test were analyzed by gas chromatography. Parathion (as well as the other insecticides studied) moved downward more easily in the sand than in the loam, and was strongly retained in the humus covering. No parathion was found in any of the soil or water samples after 8 months.

In a follow-on test, 10 ml of concentrated parathion was applied to the center of the lysimeter surfaces. For an additional period of 15 months, one-half of the lysimeters were again watered with the equivalent of normal rainfall, the other half at the excessive rate, equivalent 0.4 in. of precipitation each day. After 15 months in this second test, very little parathion was found at the 50- to 100-cm soil levels.

Sethunathan (1973) studied the persistence of parathion in India in five acid soils under flooded conditions. The product degraded faster in soils which had a high organic matter content. The fastest degradation occurred in an acid sulfate soil with an organic content of 12.2%. The author attributes this to microbial participation. Hydrolysis of parathion to _p_-nitrophenol was enhanced by repeated additions of parathion to an alluvial soil. Heat treatment of this system retarded the rate of hydrolisis of parathion, indicating the role of biological agents in the process. A _Bacillus_ species capable of readily decomposing _p_-nitrophenol was isolated from the parathion enriched flooded alluvial soil.

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[1] Herzel, F., "The Behavior of Several Persistent Insecticides in the Soil," _Bundesgesundheitsblatt_ 14(3):23-28 (1971).

Sethunathan and Yoshida (1973)[1] studied the persistence and degradation of parathion in Philippine rice soils in the laboratory, simulating submerged and upland conditions. Parathion was incubated in the different soils at 30°C, then extracted with a hexane-acetone mixture and determined by gas chromatography. Parathion disappeared more rapidly from submerged soils than from upland soils. In submerged soils, it was reduced to aminoparathion. Autoclaving of the soils increased the persistence of parathion under submerged conditions, indicating microbial involvement in the degradation. Inoculation of flooded soils with *Flavobacterium* species accelerated the rate of degradation of parathion as compared to uninoculated soil. This bacterium, isolated from paddy water, had been shown to be highly efficient in hydrolyzing parathion in previous tests.

Chopra and Khullar (1971)[2] found that the following experimental conditions accelerated the rate of degradation of parathion in soils in laboratory tests: increased concentration of parathion; increased temperatures; increased time of exposure to UV light; increased relative humidity; and increased pH. Hydrolysis was more rapid in alkaline and acidic soils than in neutral ones. The authors point out that some of the loss occurring with increasing temperature may have been due to vaporization.

Swoboda and Thomas (1968)[3] investigated the movement of parathion in eight important agricultural soils from various locations in Texas in laboratory studies. ^{32}P-labeled parathion was applied at an initial concentration of 16.5 ppm (equivalent to 66 lb AI/acre-foot) to laboratory columns prepared from the soils, and leached through the columns with distilled water. Distribution coefficients for the adsorption of parathion were calculated, and the amount of rainfall required to leach parathion to a depth of 60 in. was estimated. This amount varied from 230 in. for a Nacogdoches clay subsoil to 1,725 in. for a Houston Black clay surface soil. Desorption experiments indicate that parathion is not effectively displaced by inorganic electrolyte salts, or by aqueous solutions. However, it moved swiftly through the soil columns when leached with ethanol. From the data obtained, the authors conclude that it is very unlikely that parathion could contaminate underground water supplies beneath any of these soils by leaching under normal rainfall conditions.

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[1] Sethunathan, N., and T. Yoshida, "Parathion Degradation in Submerged Rice Soils in the Philippines," J. Agr. Food Chem., 21(3):504-506 (1973).

[2] Chopra, S. L., and F. C. Khullar, "Degradation of Parathion in Soils," J. Indian So. Soil Sci., 19(1):79-85 (1971).

[3] Swoboda, A. R., and G. W. Thomas, "Movement of Parathion in Soil Columns," J. Agr. Food Chem., 16(6):923-927 (November/December 1968).

Leenheer and Ahlrichs (1971)[1/] performed kinetic and equilibrium studies on the adsorption of parathion on soil organic matter surfaces, using two types of Indiana soil, i.e., a silty clay loam and a muck soil. They found that the differences in adsorptive characteristics of various types of soil organic matter were small, but changing the saturating cation from calcium to hydrogen greatly increased the adsorptive capacity for parathion. The authors conclude that the fast adsorption rates, the low heats of adsorption, the reversibility of adsorption, and the high adsorptive capacities on hydrophobic surfaces tend to rule out chemisorption, pointing instead to physical adsorption with formation of Van der Waals bonds between the hydrophobic portions of the pesticide molecules and the adsorbent surfaces in aqueous systems.

Saltzman et al. (1972),[2/] Saltzman and Yaron (1972)[3/], and Yaron and Saltzman (1972)[4/] used parathion labeled with ^{14}C in the alkyl chain in studies on the influence of soil organic matter on the adsorption of parathion. In aqueous solutions, parathion had greater affinity for organic than for mineral colloid adsorptive surfaces. Parathion bonding was stronger on organic than on mineral surfaces. When aqueous and hexane solutions containing from 1 to 10 μg of parathion per milliliter were applied to three soil types under different conditions, there was no appreciable thermic effect on the adsorption of parathion from aqueous solutions after correction for increased solubility. Increasing soil water content resulted in decreasing parathion adsorption.

[1/] Leenheer, J. A., and J. L. Ahlrichs, "A Kinetic and Equilibrium Study of the Adsorption of Carbaryl and Parathion Upon Soil Organic Matter Surfaces," Soil Sci. Am. Proc., 35(5):700-705 (1971).

[2/] Saltzman, S., L. Kliger, and B. Yaron, "Adsorption-Desorption of Parathion as Affected by Soil Organic Matter," J. Agr. Food Chem., 20(6):1224-1226 (1972).

[3/] Saltzman, S., B. Yaron, "Parathion Adsorption from Aqueous Solutions as Influenced by Soil Components." In: Fate of Pesticides in Environment, Gordon and Breach, London, pp. 87-100 (1972).

[4/] Yaron, B., and S. Saltzman, "Influence of Water and Temperature on Adsorption of Parathion by Soils," Soil Sci. Soc. Am. Proc., 36(4):583-586 (1972).

In another test series, adsorption isotherms were obtained for 20 soils, Na-kaolinite, Na-montmorillonite, organic material, and glass beads. The results showed that the main soil components affecting parathion adsorption are the expanding-lattice clays and the organic matter which present linear adsorption isotherms. Parathion adsorption on nonexpanding-lattice clays and on noncolloidal soil materials is reduced.

King and McCarty (1968)[1] developed a chromatographic model for predicting pesticide migration in soils. For several insecticides, including parathion, theoretical elution curves based on chromatographic theory were developed and compared with experimental degradation and leaching data. Four soil types, and different column lengths and pesticide application rates were employed. Analysis of the experimental data and their correlation with the theoretically derived curves establish the feasibility of predicting the extent to which organic phosphate insecticides may be leached through soil columns. The equation describing the chromatographic movement of the pesticides takes into account the noncontinuous flow conditions which would be expected in typical agricultural practice. Critical variables include the degradation rate of the pesticide, the distribution of the pesticide in the soil water environment, and the decay reaction rate constant under the environmental conditions prevailing. Problems may arise when the water solubility of the pesticide is approached, if the degradation of the pesticide increases due to biological acclimation, and from similar factors.

Field and Combined Field-Laboratory Studies -

Short-term studies - Several authors have studied the persistence of parathion under field conditions.

Lichtenstein and Schulz (1964)[2] applied parathion at 5 lb AI/acre to Carrington silt loam field plots. Residue levels of parathion of approximately 0.1 ppm (3.1% of the applied dosage) were reached under field conditions within 90 days after the insecticidal application to the soil.

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[1] King, P., and P. L. McCarty, "A Chromatographic Model for Predicting Pesticide Migration in Soils," Soil Sci., 106(4):248-261 (1968).

[2] Lichtenstein, E. P., and K. R. Schulz, "The Effects of Moisture and Microorganisms on the Persistence and Metabolism of Some Organophosphorus Insecticides in Soils, with Special Emphasis on Parathion," J. Econ. Entomol., 57:618-627 (1964).

Under laboratory conditions (30°C), 30% of the applied parathion dose was lost within 12 days after application to a loam soil. In further laboratory studies, the effects of moisture and microorganisms on the persistence and metabolism of parathion in Carrington silt loam soil were tested at 30°C. Parathion was most persistent in dry soil, least persistent in soils with high moisture content. There was no loss of parathion through volatilization. Parathion was degraded either by hydrolysis or by reduction to its amino form, depending on populations of soil microorganisms. In autoclaved soils that had low numbers of microorganisms, or in dry soils in which microorganism activity was low, parathion residues persisted for a relatively long time. No aminoparathion was formed in autoclaved soils. Paraoxon, a parathion metabolite, was hydrolyzed within 12 hr after its application to loam soil at 20 ppm. During that time, the amount of paraoxon decreased constantly, while the amount of *p*-nitrophenol increased. Aminoparathion or *p*-nitrophenol, added to soils, disappeared within 2 and 16 days, respectively.

Burkhardt and Fairchild (1967b)[1] applied parathion (from a granular formulation) at 1, 2 and 4 lb AI/acre in a 7-in. band at planting time in field studies located in four counties in Missouri. Soil samples were taken at various depths both in the treated row and adjacent to the row, and bioassayed with 21-day old house crickets, *Acheta domesticus*, to determine bioactivity and vertical and lateral movement of the insecticide. Based on the bioassay results from all field plots combined, parathion at 1 lb AI/acre provided 78% mortality after 1 day; 77% after 1 week; 86% after 2 weeks; 47% after 4 weeks; 3% after 6 weeks; 0% after 8 weeks. At the 4-lb AI/acre rate, parathion provided 100% mortality during the first 2 weeks after application; 98% after 4 weeks; 33% after 6 weeks; 25% after 8 weeks. Results from the 2-lb AI/acre rate were intermediate. At all three application rates, there was a sharp drop in bioactivity after the 4th week. There was little downward movement of parathion; in the majority of soil samples from the parathion-treated plots, very little or no bioactivity was found more than 3 in. below the soil surface. Horizontal movement of parathion likewise was minimal.

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[1] Burkhardt, C. C., and M. L. Fairchild, "Bioassay of Field-Treated Soils to Determine Bioactivity and Movement of Insecticides," *J. Econ. Entomol.*, 60(6):L1602-1610 (1967b).

Sacher et al. (1972)[1] studied the bioactivity and persistence of several parathion formulations in the soil under laboratory and field conditions. They found that moisture and soil type influenced the rate of degradation of parathion, but this influence was mediated primarily by the activity of microorganisms. Highest biological activity against larvae of the Western corn rootworm, Diabrotica virgifera, in soil was obtained with a low-concentrate formulation with minimum binding of parathion (1% kaolinite dust, insect LD_{50} 0.05 ppm), whereas low activity resulted from a tightly bound, concentrated, thinly distributed formulation (10% on charcoal, LD_{50} 6.0 ppm). However, parathion applied by way of the kaolinite dust formulation had a half-life in soil of only 6 days, compared to more than 72 days for the 10% charcoal granules.

Microbial degradation appeared to be a major contributor to parathion breakdown in soil. Ray silt loam was treated with parathion 10% granules on vermiculite at 20 ppm. Only about 30% of the applied rate was still present, as determined by chemical assay, 6 weeks after treatment. When the same soil was sterilized by treatment with methyl bromide (1 g/100 g of soil) prior to the parathion application, there was no appreciable parathion decline after 6 weeks.

The importance of microbial activity in the soil degradation of parathion was further demonstrated in field tests in which it was shown that the rate of parathion degradation was much slower in field soils in the winter months when soil microorganisms are dormant. Most rapid breakdown occurred in August and September when the half-life of parathion in the soil was as short as 1.5 weeks.

Knutson et al. (1971)[2] studied the relationships between insecticide usage and resulting residues in a newly developed irrigation district in central Kansas. This area had previously been under dry land farming practices with little exposure to insecticides. Parathion was applied to irrigated corn at planting time at treatment rates ranging from 0.79 to 1.40 lb AI/acre each year during a 4-year period, 1966 to 1969. Soil samples taken immediately after each annual application had average parathion residues ranging from 0.5 ppm to 4.07 ppm. No detectable parathion residues were found in samples obtained 1.5 and 2.5 months after treatment. No residues were detected in corn grain, in capped wells from 13 to 71 ft deep, nor in surface water samples from the Smoky Hill River and the Cedar Bluff Reservoir, the water bodies draining the area.

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[1] Sacher, R. M., G. F. Ludvik, and J. M. Deming, "Bioactivity and Persistence of Some Parathion Formulations in Soil," J. Econ. Entomol., 65(2):329-332 (April 1972).

[2] Knutson, H., A. M. Kadoum, T. L. Hopkins, G. F. Swoyer, and T. L. Harvey, "Insecticide Usage and Residues in a Newly Developed Great Plains Irrigation District," Pest. Monit. J., 5:17-27 (1971).

Harris and Sans (1971)[1/] studied insecticide residues in soils on 16 farms in southwestern Ontario in 1964, 1966, and 1969. Residues of organophosphate insecticides were determined qualitatively by a nonspecific enzymatic analysis, and quantitatively by GLC where possible. The use pattern of insecticides in the area showed that organophosphate insecticides were used at an increasing rate from 1965 to 1969. Preliminary findings indicated that the more extensive use of the organophosphate insecticides resulted in residues of these products in vegetable soils, while the enzyme inhibition tests in 1969 indicated that inhibitory substances were generally below significant levels in field crop, tobacco, and orchard soils. Parathion and paraoxon residues were positively identified by GLC analysis on at least one of the 16 farms studied.

Mol et al. (1972)[2/] investigated the persistence of parathion residues in the field after winter applications of parathion at 3.0 and 6.0 lb/acre in Australia. Parathion residues in top soil decreased from 6.5 ppm initially to 4.7 ppm 3 months after application of the lower rate, and from 10.7 ppm to 8.5 ppm at the higher rate. No detectable residues were found in the soil at either application rate after 8 months. Little soil penetration of parathion was found below a depth of 1 in.

Kilgore et al. (1972)[3/] analyzed soil samples from a vineyard sprayed with parathion emulsifiable concentrate at 4 and 8 lb. AI/acre for parathion residues. Following the 4 lb/acre application, parathion

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[1/] Harris, C. R., and W. W. Sans, "Insecticide Residues in Soils on 16 Farms in Southwestern Ontario - 1964, 1966, 1969," Pest. Monit. J., 5(3):259-267 (December 1971).

[2/] Mol, J. C. M., D. L. Harrison, and R. H. Talfer, "Parathion: Toxicity to Sheep and Persistence on Pasture and in Soil," N.Z. J. Agr. Res., 15(2):306-320 (1972).

[3/] Kilgore, W. W., N. Marey, W. Winterlin, "Parathion in Plant Tissues: New Considerations." In: Degradation of Synthetic Organic Molecules in the Biosphere, National Academy of Sciences, pp. 291-312 (1972).

concentration in the soil was 21 ppm at 0 days, 0.06 ppm at 46 days after treatment. Following the 8-lb/acre treatment, parathion concentration in the soil was 70 ppm at 0 days, 0.13 ppm at 46 days after treatment.

Long-term field studies - Stewart et al. (1971)[1] and Chisholm and MacPhee (1972)[2] investigated the long-term persistence and effects of parathion (and several other pesticides) in the soil in a field experiment at Kentville, Nova Scotia. Parathion was applied to a sandy loam at the rate of 31.4 lb AI/acre (equivalent to 15.7 ppm in the upper 6 in. of soil) from a 15% wettable powder formulation. This rate was intended to approximate the maximum concentration in the soil (neglecting loss through volatilization, etc.) that might accrue from an intensive spray program carried on over a 20- to 25-year period, and it was applied each spring from 1949 to 1953 inclusive. Each time, the insecticide was thoroughly incorporated to a depth of 6 in. with a rotary cultivator, and various crops were grown on the plots each year up to 1969. In the spring of 1969, 16 years after the last application, soil samples were taken at various depths from the experimental plots and analyzed for parathion residues. The 0.06 ppm of parathion was found in the 0-to 4-in. soil layer; 0.063 ppm in the 4-to 8-in. layer, 0.008 ppm in the 8-to 12-in. layer, and a trace amount at the 12-to 16-in. depth. Thus, about 0.1% of the total parathion applied to the plot (157 lb AI/acre) was still present 16 years after the last application.

Voerman and Besemer (1970)[3] reported on an experiment started in 1953 at Wageningen, The Netherlands, and continued for 15 years, up to 1968. Parathion (and other insecticides) were sprayed in two concentrations on crops several times each year. In addition, soil treatments of these insecticides were applied once a year. Soil and crop samples were taken and analyzed throughout the entire period. The experiment was conducted in light, sandy soil. From 1963 to 1968, the total quantities of parathion applied to three different plots were 10.4, 21.5 and 111.4 lb AI/acre. Soil residues found in the 0-to 4-in.

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[1] Stewart, D. K. R., D. Chisholm, and M. T. H. Ragab, "Long-Term Persistence of Parathion in Soil," Nature, 229:47 (1971).

[2] Chisholm, D., and A. W. MacPhee, "Persistence and Effects of Some Some Pesticides in Soil," J. Econ. Entomol., 65(4): 1010-1013 (August 1972).

[3] Voerman, S., and A. F. H. Besemer, "Residues of Dieldrin, Lindane, DDT, and Parathion in a Light Sandy Soil After Repeated Application Throughout a Period of 15 Years," J. Agr. Food Chem., 18(4): 717-719 (1970).

soil layer after 15 years of repeated applications were 0.01, 0.02 and 0.06 ppm, respectively, equivalent to 0.1% of the applied amount for each application rate. No parathion residues were detected in the 4- to 8-in. soil layer in the plots that had received the two lower rates, while 0.02 ppm was found in that layer following the heaviest parathion applications. No parathion residues were found in deeper soil layers.

Massive soil contamination with parathion may result from the spillage of pesticides, especially from repeated spillage in areas where spray tanks are routinely loaded. Wolfe and Durham (1966)[1] set up a replicated field test to simulate spillage of undiluted parathion emulsifiable concentrate containing 45.6% AI/gal. In another test, soil contamination from tank drainage on the ground with dilute (1x) and concentrate (8x) liquid sprays, using both wettable powder and emulsifiable concentrate formulations, was simulated. The test plots were in orchard silt-loam soil with high organic material content, and a pH ranging from 4.4 to 5.5. The plots were exposed to weathering and regular sprinkler irrigation.

The top 1 in. of soil contaminated with the emulsifiable concentrate contained 59,000 ppm of parathion initially. Forty-six percent of this residue was still present after 2 years. The soil contaminated with the dilute formulations had initial surface-soil concentrations of parathion of 98 to 108 ppm from the 1x spray, and from 370 to 1,126 ppm from the 8x spray.

The authors then collected and analyzed top soil samples from 37 spray tank fill sites throughout the Wenatchee Valley, Washington area. The time intervals between contamination of soil and sampling for analysis were not known. Parathion residues found ranged from 0.3 to 330 ppm, with a mean of 48.4 ppm. The authors conclude that parathion residues may persist in the soil at relatively high levels for long periods of time.

The experimentally contaminated field plots described by Wolfe and Durham (1966) were monitored further, and more recent findings were reported by Wolfe et al. (1973). Parathion residues persisted at relatively high levels for 5 years following the initial applications.

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[1] Wolfe, H. R., and W. F. Durham, "Spillage of Pesticides and Residues in Soil," Wash. State Hort. Assoc. Proc., 62:91-92 (1966).

The levels of parathion in the soil had declined considerably by the end of the first year. The rate of disappearance was much slower the second year, and the decline curves indicate very little further disappearance of residues beyond the third year. Six years after application of the 45.6% concentrates, parathion residues ranging from 9,330 to 15,370 ppm were still present in the upper 3 in. of soil. Residues found in the 3-to 9-in. soil layer ranged from 9.48 to 9,900 ppm, while concentrations ranging from 0.1 ppm to trace amounts were found in five different layers of soil ranging from 9 to 24 in. in depth. Residues from the dilute sprays disappeared more rapidly. Where the 1x (0.03%) concentration had been applied, average parathion levels after 5 years ranged from 0.6 to 1.2 ppm in the top 1 in. of soil, and from 0.2 to 0.3 ppm in the 1- to 3-in. depth. Where the 8x (0.24%) concentration was applied, average residue levels after 5 years were 0.4 to 1.5 ppm in the top 1 in. of soil, and from 1.2 to 3.60 ppm in the 1- to 3-in. level.

Monitoring studies - In the National Soils Monitoring Program for pesticides, 1,729 samples of cropland soils from 43 states were collected in 1969 (Wiersma et al., 1972b).[1] Of these, 66 samples were analyzed for organic phosphate residues, and seven of the 66 (10.6%) contained parathion residues ranging from 0.01 to 3.01 ppm, the mean residue level being 0.06 ppm. One hundred ninety-nine samples of noncropland soil were also obtained, but none of these were analyzed for organophosphate residues.

In the National Soils Monitoring Program for Pesticides in 1970 (Crockett et al., 1970)[2] soil and crop samples were collected from 1,506 cropland sites in 35 states. Pesticide use records indicated that parathion was used at 20 of 1,346 sites sampled, that is, 1.49% of all sites. No analyses of soil samples for parathion residues are reported. Samples of alfalfa, field corn kernels, cotton stalks and green bolls, grass hay, field corn stalks, cotton seeds, mixed hay, and soybeans (beans) were analyzed for organophosphate residues.

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[1] Wiersma, G. B., H. Tai, and P. F. Sand, "Pesticide Residue Levels in Soils, FY 1969 - National Soils Monitoring Program," Pest. Monit. J., 6(3):194-228 (December 1972b).

[2] Crockett, A. B., G. B. Wiersma, H. Tai, W. G. Mitchell and P. J. Sand, "National Soils Monitoring Program for Pesticide Residues - FY 1970," U.S. Environmental Protection Agency, Technical Services Division, unpublished manuscript (1970).

Parathion residues found were nondetectable in most instances; in some, their mean concentration was 0.01 ppm or less.

Wiersma et al. (1972a)[1] monitored pesticide residues in commercially grown onions and in the soil on which these onions were grown in 1969. A total of 76 sites in 10 major onion producing states were sampled. According to pesticide use records, parathion had been used on 34.4% (36.4%) of the farms sampled, at an average rate of 1.64 lb AI/acre. However, parathion residues (ranging from 0.02 to 2.55 ppm, averaging 0.22 ppm) were found in 51.5% of the soil samples. In most instances, the average amount of residue detected was considerably less than the amount applied when converted to pounds AI per acre. Despite the high residue levels in the soils, no residues of parathion (or of any of the other pesticides) were detected in the onion samples.

Stevens et al. (1970)[2] reported on a pilot study conducted nationwide at 51 locations in 1965, 1966, and 1967 to determine pesticide residue levels in soil. Samples were collected from 17 areas in which pesticides are used regularly, 16 areas with a record of at least one pesticide application, and 18 areas with no history of pesticide use. This study was apparently aimed primarily at persistent chlorinated hydrocarbon pesticides; only those organophosphates that were amenable to chlorinated pesticide clean-up methods were detected, and no serious attempts were made to quantitate metabolites or oxygen analogs of the organophosphates. Pesticide use records indicated that parathion had been used at a number of the sites sampled, but only one single detection of parathion is reported, i.e., a residue of 0.06 ppm in one of seven fields sampled near Tulelake, California.

The data on parathion soil residues reported in the foregoing four reports emanating from the National Soils Monitoring Program for Pesticides are subject to question because in this program, soil samples are being shipped and stored at room temperature until processed for analysis, as reported by Stevens et al. (1970), and confirmed in a recent personal communication to this reviewer by G. B. Wiersma. No information is given in these reports on the relationships between time of pesticide application, time of sampling, and time of processing and analysis of the samples. No information is available on the effects of shipping and storage of the samples on the parathion residues that may have been present at the time of sampling.

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[1] Wiersma, G. B., W. G. Mitchell, and C. L. Stanford, "Pesticide Residues in Onions and Soil - 1969," Pest. Monit. J., 5(4): 345-347 (March 1972a).

[2] Stevens, L. J., C. W. Collier, and D. W. Woodham, "Monitoring Pesticides in Soils from Areas of Regular, Limited, and No Pesticide Use," Pest. Monit. J., 4(3):145-164 (December 1970).

Applegate (1970)[1] collected samples of soil, vegetation, birds, rodents, and lizards from a large area of the Big Bend National Park, Texas, and analyzed them for insecticide residues. Samples were placed in an ice chest as quickly as possible after collection, maintained under refrigeration, then taken to Presidio, Texas, 3 to 5 days later where they were placed in deep freezers and held for 10 to 14 days in frozen storage until processing and analysis. Parathion residues were found in 1 out of 9 samples of surface soil (0.01 ppm); in 1 out of 9 samples of leatherstem, Jatropha dioica (0.01 ppm); in 2 out of 20 samples of muscle tissue of rodents (0.04, 0.11 ppm); in 3 out of 19 samples of whole lizards (0.01, 0.01, 0.10 ppm); and in 6 out of 19 samples of bird muscle (0.01 to 0.21 ppm). There were no known direct applicators of parathion in the park. The nearest areas using insecticides are all south of the park in Mexico where parathion and other pesticides have been applied routinely to cotton fields. It is not known how the specimens that were found to contain parathion residues acquired these. The purpose of this study was to establish a baseline for future investigations to show possible changes in the level of pesticide pollution of this park.

The California Department of Water Resources (1969, 1970)[2,3] reported on pesticide concentrations in surface and subsurface drain effluents in the San Joaquin Valley. In 1969, 14 samples of surface drain effluents were analyzed for organophosphorus compounds. Parathion was detected in three of these, at concentrations ranging from 6 to 500 ppt (parts per trillion), averaging 37 ppt in all samples analyzed, 173 ppt in the positive samples. In the same year (1969), 41 samples of subsurface drain effluents were analyzed for organophosphate compounds. Residues were detected in 19 of these, but no identification was made of specific pesticides involved.

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[1] Applegate, H. G., "Insecticides in the Big Bend National Park," Pest. Monit. J., 4(1):2-7 (1970).

[2] California Department of Water Resources, San Joaquin Valley Drainage Monitoring Program, 1969 Summary, Sacramento, California (1969). (In: Li and Fleck, op. cit., 1972.)

[3] California Department of Water Resources, San Joaquin Valley Drainage Monitoring Program, 1970 Summary, Sacramento, California (1970). (In: Li and Fleck, op. cit., 1972.)

In 1970, 18 samples of surface drain effluents were analyzed for organophosphate compounds. Parathion was positively identified in one of these; the concentration found was 190 ppt. Of 60 samples of subsurface drain effluents analyzed for organophosphates, 10 were found to contain residues of undetermined members of this group.

These data show that only very small quantities of parathion and of other organophosphate insecticides were present in these effluents. The concentrations of these pesticides were somewhat higher in the surface than in the subsurface drains. These findings are in good agreement with those of other authors, indicating that organic phosphates do not leach appreciably through soil profiles.

The results of the 1972 National Soils Monitoring Program have not yet been published, and a computer printout of the analytical data byproducts is not expected to become available until later this summer. Thus, data from the program is not included in this review.

Summary - Scientific data on the reviewed laboratory, field and monitoring studies on residues and fate of parathion show that the persistence of this chemical in the soil varies considerably, depending on a number of factors. Parathion residues in the soil degrade by chemical hydrolysis as well as by microbial action. Parathion is strongly adsorbed by organic matter. Thus adsorbed, the chemical becomes unavailable for insecticidal action, and appears to be protected to a degree against degradation. No data are available on the fate of such adsorbed parathion residues; it is not known whether they eventually degrade, or if the under what conditions they might become reactivated by desorption. No information appears to be available on the fate of the first degradation products, especially *p*- nitrophenol and aminoparathion, or regarding effects on organisms other than mammals and insects.

Parathion soil residues resulting from crop protection uses at recommended dosage levels are apparently degraded in the soil, in most cases, within a few weeks or months after application. However, small fractions of originally applied quantities may persist in the soil for prolonged periods of time. In the case of heavy soil contamination, parathion soil residues have persisted for at least 16 years.

Parathion degradation in the soil appears to be temperature-dependent and proceeds much more slowly in colder climates. Thus, Canadian investigators and one researcher from Australia who studied winter application of parathion reported much slower decline rates than were experienced in California.

Monitoring studies in which samples to be analyzed for parathion residues are not frozen promptly after collection are of questionable value in determining environmental residues of this pesticide. In such studies, the time(s) and rate(s) of application in relation to time of sampling should also be reported.

Residues in Water

Laboratory and Field Studies - Studies dealing with the fate of parathion residues in water are discussed in this section in approximate chronological order.

Lichtenstein et al. (1966)[1] studied the toxicity of parathion and several other insecticides in water to mosquito larvae after percolation of the water through insecticide-contaminated agricultural loam soil. In distilled water, parathion caused 85% mortality of mosquito larvae within 24 hr at 0.035 ppm. When water was percolated through soil treated with parathion at the rate of 50 ppm, parathion residues were present in the water as determined by gas liquid chromatography and 100% mosquito mortality. Parathion was also present in water percolated through soil treated at the rate of 1 ppm, when percolation took place on the day of treatment. The 0.013 ppm of parathion was found analytically in the percolated water. Seventeen days after soil treatment, the parathion residue in the soil was 0.089 ppm; in the percolated water 0.001 ppm. The percolated water was not toxic to mosquito larvae at this time. The authors conclude that the contamination of water by insecticidal soil residues is to a large degree a function of the water solubility of a particular insecticide. Insecticidal concentration in the soil and specific properties of a given pesticide also are important.

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[1] Lichtenstein, E. P., "Increase of Persistence and Toxicity of Parathion and Diazinon in Soils with Detergents," J. Econ. Entomol., 59(4):985-993 (1966).

Graetz et al. (1970)[1] investigated the pathways and mechanisms of parathion degradation in lake sediments. Lake sediment samples were taken from two lakes in Wisconsin, and the mechanisms and rates of parathion degradation were determined in dry-heat-sterilized lake sediment, in aerobic and anaerobic peptone solutions in a peptone enriched sediment system, and in intact sediments from the two lakes. Parathion was readily susceptible to microbial degradation in aerobic and anaerobic environments. Reduction of the nitro group to form aminoparathion was a major microbial degradation pathway. Under aerobic, but not under anaerobic conditions aminoparathion was degraded further, apparently by another microbial process. Parathion degradation was slower in a slightly acid sediment than in a calcareous one. The authors conclude that the persistence of parathion in the natural aquatic environment is affected markedly by microbial activity. Without microbial activity, parathion would remain in the environment for several months, while in biologically active (aerobic or anaerobic) environments, it would be degraded in a matter of weeks.

Eichelberger and Lichtenberg (1971)[2] investigated the persistence of parathion and a number of other common pesticides in raw river water over an 8-week period. Aliquots of 10 μg/liter of parathion from a freshly prepared 0.1% solution in acetone were injected into samples of raw water from the Little Miami River, a relatively small stream receiving domestic and industrial wastes and farm runoff. The dosed raw river water was kept in the laboratory in closed glass containers at room temperature, exposed to natural and artificial light. The same concentration of parathion was also added to distilled water in the same manner. In river water (alkalinity 98 μg/liter) 50% of the original concentration of parathion remained after 1 week, 30% after 2 weeks, less than 5% after 4 weeks. With the use of thin layer chromatography, it was observed that parathion was hydrolyzed to *p*-nitrophenol and diethylthiophosphoric acid. Parathion remained stable in distilled water for 3 weeks.

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[1] Graetz, D. A., G. Chesters, T. C. Daniel, L. W. Newland, and G. B. Lee, "Parathion Degradation in Lake Sediments," *J. Water Poll. Control Fed.*, 42 (2, Pt. 2):R76-R94 (1970).

[2] Eichelberger, J. W., and J. J. Lichtenberg, "Persistence of Pesticides in River Water," *Environ. Sci. Tech.*, 5(6):541-544 (1971).

Miller et al. (1966) investigated the persistence and movement of parathion in cranberry bog irrigation waters. Irrigation water from a cranberry bog that had been treated with parathion at a rate equal to 1 lb AI/acre through overhead sprinkler irrigation was sampled immediately after application, and every 24 hr thereafter for a period of 96 hr. The same experiment was repeated 14 days later. The authors report data representing the mean of the two experiments. Samples collected from an adjacent irrigation ditch contained 750 ppb of parathion immediately after application; 60 ppb after 24 hr; 25 ppb after 48 hr; 10 ppb after 72 hr; and 5 ppb after 96 hr. In an associated drainage canal, 30 ppb of parathion were found immediately after application, indicating water seepage through a flood gate. When this canal was resampled after 24 hr, 3.0 ppb of parathion were detected. Twenty-four hours after the application (but at no other time period), trace amounts of parathion were also detected at two locations 50 and 150 yd from the application site in the drainage canal down from the point where seeping irrigation water would enter the canal waters.

Schaefer and Dupras (1969)[1/] studied the effects of water quality, temperature and light on the stability of parathion used for mosquito control. Samples of pasture water from pastures with a history of mosquito control difficulties in alkaline soils in two California counties were collected and passed through a coarse filter to remove larvae and large pieces of organic matter. The 150-ml samples of pasture water and distilled water in glass jars were treated with parathion from a 0.15-µg/ml acetone solution to give a concentration of 0.1 ppm of the insecticide, the concentration normally encountered in pasture water following application of 0.1 lb AI/acre. The jars were moved to the field and placed into pasture water to expose them to the same conditions of color, temperature and natural solar radiation as field pasture water. Both pasture and distilled water samples were extracted after 0, 2, 4, and 8 hr of exposure. Parallel tests were carried out in the laboratory where samples were kept in the dark and at constant temperatures of 24 and 38°C. Water quality had little, and light had no, effect on the stability of parathion. This insecticide was very stable both under field and laboratory conditions and was only slightly influenced by temperature. After 8 hr, there was 10% more loss of AI at 38 than at 24°C.

[1/] Schaefer, C. H., and E. Dupras, Jr., "The Effects of Water Quality, Temperature and Light on the Stability of Organophosphorus Larvicides Used for Mosquito Control," Proc. Papers Ann. Conf. California Mosquito Control Assoc., 37:67-75 (1969).

Paris and Lewis (1973)[1] recently presented a rather brief, but fairly complete review of the current extent of knowledge on the chemical and microbial degradation of 10 selected pesticides, including parathion, in aquatic systems. They briefly cite many of the research studies reviewed in greater detail in this report, but do not draw any conclusions from their overview of the parathion data.

Several authors from abroad have contributed data on parathion in aquatic systems. Greve (1972)[2] identified parathion residues in Rhine water at 0.01 to 0.1 ppb. Quentin (1972a, 1972b)[3,4] dealt with the analytical, decontamination, and hygienic problems associated with the presence of residues of parathion and other pesticides in water, including drinking water supplies. Adoption of a reduced maximum allowable concentration of parathion is being urged in West Germany. Pesticide contamination of surface waters must be monitored more closely in the future as the use of surface water for drinking water purposes increases. The author emphasizes that maximum allowable concentrations of single pesticides may be inadequate when several pesticides are present in the same water.

Manescu (1971)[5] observed a decreased rate of oxygen consumption by the self-purifying bacterial flora in surface water samples following treatment with 100 mg/liter of parathion. Oxygen consumption was determined by the micromanometric Warburg procedure. Compared to other pollutants, metals, cationic detergents, or chlorine, pesticides ranked low in their inhibitory action on the oxygen consumption of the bacteria.

The reports reviewed in this section indicate, and the recent literature search by Paris and Lewis (1973) confirms, that relatively little information is available on the persistence and fate of parathion residues in water, especially under actual field conditions.

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[1] Paris, D. F., and D. L. Lewis, "Chemical and Microbial Degradation of Ten Selected Pesticides in Aquatic Systems," Residue Rev., 45:95-124 (1973).

[2] Greve, P. A., "Toxic Organic Trace Pollutants in Surface Water," Chem. Weekblad. 41(68):11, 13, 15 (1972).

[3] Quentin, K. E., "Pesticides in Water - Determination, Removal, and Limiting Values," Chem. Ing. Tech., 44(20):1172-1176 (1972a).

[4] Quentin, K. E., "Evaluation and Significance of Residue Values from the Viewpoint of the Water Biocenosis and Drinking Water Quality," Schriftenr. Ver. Wasser-Boden-Lufthyg., 34:19-28 (1972b).

[5] Manescu, S., "The Value of the Manometric Procedure in Studying the Chemical Micropollutant Action Potential on the Self-Purifying Water Flora," Igiena 20(5):275-286 (1971).

Monitoring Studies - Dupuy and Schulze (1972)[1] and Achulze et al. (1973)[2] reported a maximum of 1 ppb parathion in routine water monitoring. Butler (unpublished)[3] found a maximum of 170 ppb parathion in several marine fish along the Atlantic and Gulf coasts.

Residues in Air

There is little information available on the origin, presence and persistence of parathion residues in air.

Harris and Lichtenstein (1961)[4] exposed cages vinegar flies (*Drosophila melanogaster*) and houseflies (*Musca domestica*) to vapors from soils that had been treated with parathion in the laboratory or in the field. In the laboratory tests, quartz was treated at the rate of 4 ppm of parathion. There was no mortality of the insects that were kept in screened cages above the parathion-treated sant for periods of 6 or 24 hr. Under the same conditions, 100% mortality was obtained with several other insecticides, demonstrating the method's validity. Lichtenstein and Schulz (1970)[5] investigated the volatilization of parathion and other insecticides from eight different substrates. It was generally assumed that low water solubility and a relatively high vapor pressure would enhance volatilization from aqueous substrates. However, no direct relationships were established by experimental results between water solubility of a particular compound and its volatilization. Glass beads, tap water, soil water, algae in nutrient media, buffer solution, buffer solution plus 0.1% detergent, silt loam and silt loam containing 0.1% detergent were treated with ^{14}C-labeled parathion. The material volatilized within 24 hr was collected in a vapor trap and determined. The volatilization rate of parathion was very low, ranging from 0.07% of the applied dose from the soil with or without the detergent, to 1.67% of the applied does from the buffer solution containing detergent. Volatilization rates from the other substrates were intermediate between these two extremes.

Sandi (1958)[6] studied the effects of direct sunlight compared to dark conditions on parathion in a 2×10^{-3} M solution in a barbiturate buffer at pH 8.0. The results observed by two different analytical methods show that parathion is photochemically reduced, presumably to the corresponding *p*-amino compound.

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[1] Dupuy, A. J., and J. A. Schulze, Selected Water-Quality Records for Texas Surface Waters, 1970 Water Year. Texas Water Development Board, Report 149, Austin, Texas (1972).

[2] Schulze, J. A., D. B. Manigold and F. L. Andrews, "Pesticides in Selected Western Streams - 1968-71, Pesticide Monitoring J., 9(2) 124-135 (1973).

[3] Butler, P. A., Unpublished report on Environmental Protection Agency National Estuarine Monitoring Program.

[4] Harris, C. R., and E. P. Lichtenstein, "Factors Affecting the Volitilization of Insecticidal Residues from Soils," J. Econ. Entomol., 54(5): 1038-1045 (1961).

[5] Lichtenstein, E. P., and K. R. Schulz, "Volatilization of Insecticides from Various Substrates," Agr. Food Chem., 18(5):814-813 (1970).

[6] Sandi, E., "Reduction of Parathion Induced by Light," Nature, 181:499 (1958).

Stanley et al. (1971)[1] conducted a pilot study to establish a system for measuring the extent of atmospheric contamination of the air by pesticides in nine localities throughout the United States. Samples were analyzed for 19 pesticides and metabolites, including parathion. Parathion was found in only one of the nine sampling locations, i.e., Orlando, Florida. At that location, 37 samples contained detectable amounts of parathion, the maximum level found being 465 ng/cu m of air. The majority of the Orlando air samples contained parathion levels ranging from about 10 to 25 ng/m^3.

Tessari and Spencer (1971)[2] analyzed air samples from human environments in the Greeley, Colorado, area for pesticide residues. Nylon chiffon cloth screens were exposed to the atmosphere for 5 days, after which residues were extracted from them with suitable solvents and determined by gas chromatographic analysis. Screens were deployed inside and outside 12 homes of men occupationally exposed to pesticides, including parathion (farmers and pesticide formulators). Samples were collected monthly for 1 year. Parathion residues were found in a surprisingly high number of cases from test screens exposed for 5 days at a time. In the positive indoor samples, parathion residues ranged from 0.03 to 9.60 $\mu g/m^2$ of filter; the means were 0.26 $\mu g/m^2$ for the farmers, 2.24 for the formulators. The authors point out that these results are surprising because "parathion is volatile and has a relatively short environmental half-life. The farmer group had little or no use of parathion, yet the occurrence of parathion in their environmental samples, especially housedust and inside air samples, was high." The outside air levels of parathion were very similar for both types of homes sampled (means 0.75 and 1.11 $\mu g/m^2$, respectively; range 0.0 to 12.92 $\mu g/m^2$).

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[1] Stanley, C. W., J. E. Barney II, M. R. Helton, and A. R. Yobs, "Measurement of Atmospheric Levels of Pesticides," Environ. Sci. Tech., 5(5):430-435 (1971).

[2] Tessari, J. D., and D. L. Spencer, "Air Sampling for Pesticides in the Human Environment," J. Assoc. Offic. Anal. Chem., 54(6): 1376-1382 (1971).

Search of the literature and of other information sources failed to yield additional data on parathion air residues. The reports reviewed above indicate that parathion residues may be present in air, but these very limited data leave open many important questions, including origin of such air residues, relationships to parathion use and handling patterns in the area, persistence and transport patterns of such residues in air, and their significance to human and environmental health.

Residues in Nontarget Plants

Search of the literature failed to yield any reports on parathion metabolism or residues in or on nontarget plants. Fukuto and Sims (1971)[1] state in a recent text on the metabolism of insecticides and fungicides: "Surprisingly few investigations have been carried out on the metabolism of parathion and related compounds in plants in spite of its wide usage on a variety of crops."

Joiner and Baetcke (1973)[2] studied the persistence of parathion on cotton and identified its photoalteration products. ^{14}C-labeled parathion was applied to cotton four times in three successive weeks in four environmental situations, i.e., environmental growth chamber, greenhouse, controlled field exposure, and open field. After 28 days, 11.2 to 15.4% of the total radioactivity applied was recovered by methanol extraction and was found to be 58 to 68% unchanged parathion. There was a constant increase in photoalteration products, paralleled by a consistent decrease in ^{14}C-parathion over time. Photoalteration products did not include any previously unreported metabolites. In this set of experiments, parathion was found to be at least seven times more persistent than previously reported on cotton, according to the authors.

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[1] Fukuto, T. R., and J. J. Sims, "Metabolism of Insecticides and Fungicides," Pest. Environ., R. White-Stevens (ed.) Vol. I, Part I, Ch. 2, pp.147-155, Marcel Dekker, New York (1971).

[2] Joiner, R. L., and K. P. Baetcke, "Parathion: Persistence on Cotton and Identification of Its Photoalteration Products," J. Agr. Food Chem., 21(3):391-396 (1973).

Kilgore et al. (1972) studied the persistence of parathion following treatment of field plots of grapevines at 2, 4 and 8 lb AI/acre. Parathion residues in bark, leaves, berries, and soil were monitored at different time periods after application. From these field experiments that were conducted in 1969 and 1971, the following conclusions were drawn: (a) parathion tends to stabilize in grape bark for at least 15 months at a level of 25 ppm; (b) no degradation of the parent compound occurs on the surface of grape plants or within the tissue after penetration; the parathion-paraoxon ratio found in the insecticide at the time of application did not change over the entire experimental period; (c) rapid loss of parathion occurs after application, mostly attributable to volatilization, which was then followed by an increasingly slower rate of disappearance; (d) parathion penetrates cane bark and reaches wood as well as pith tissues; and (e) parathion translocates via xylem into the emerging buds and the expanding new shoots.

Further tests on the absorption and translocation of parathion in roots were conducted in bean, barley and grape plants. The results obtained in this series of tests confirm reports of other authors that parathion actually penetrates and translocates in plants. It is suggested that the metabolites reported in earlier studies to be present in foliage are due to microbial activity in the soil, and that no significant modification of parathion occurs in plant tissue. Parathion disappears quite rapidly from the surface of plants after spraying. It is estimated that over 90% of the initial deposit is lost by volatilization during the first few days. The remainder is stabilized in the bark (in the case of grapevine), or penetrates into plant tissue. The amount of parathion that gains entry into plants is persistent, not subject to significant metabolic or chemical modification, translocated within plants, and to decline primarily as a result of dilution by plant growth. The authors conclude from their work and other studies that parathion appears to be much more persistent in the environment than previously believed.

Bioaccumulation, Biomagnification

Miller et al. (1966) found that mummichog (*Fundulus heteroclitus*) and mussels (*Elliptio complanatus*), in a model cranberry bog containing 0.07 ppm parathion, within 24 hours had levels of 1.68 and 0.99 ppm, respectively. The ambient level of parathion in the water at the end of 24 hours was 0.02 ppm. After 144 hours the ambient water level was 0.002 ppm, mummichog, 0.22 ppm and mussels, 0.04 ppm.

Metcalf (1972)[1] states: "Both the organophosphates and carbamates are not persistent in soil and do not accumulate in body fat."

Thus, experimental data on possible bioaccumulation and biomagnification of parathion appear to be almost nonexistent. However, the physical, chemical, and biological properties of parathion make it unlikely that biomagnification in food chains or food webs occurs, and there is no evidence that it does.

Environmental Transport Mechanisms

The data reviewed in the preceding sections of this report indicate that under field conditions, volatilization appears to be one important mechanism by which parathion may move away from the target site after application. Freed et al. (Unpublished data, quoted from von Rümker and Horay, 1972)[2] determined the propensity of parathion for volatilization and leaching under simulated field conditions for loam soils at 25°C at an annual rainfall of 59 in. (150 cm). Volatilization of pesticides under these conditions, i.e., from a porous, sorptive medium (loam soil) in a nonequilibrium situation, is different from volatilization from an inert surface or from the chemical's own surface. Therefore, the environmental volatilization index assigned to pesticides investigated in this manner may or may not parallel a chemical's vapor pressure. By this method, parathion rated a volatilization index of 3, indicating an estimated median vapor loss from treated areas of 4.45 lb/acre/year. This index number indicates a relatively high propensity for volatilization from treated fields, compared to many other pesticides.

Leaching index numbers for pesticides indicate the approximate distance that the chemical would move through the standardized loam soil profile under an annual rainfall of 59 in. (150 cm). Under these conditions, parathion rated a leaching index number of 2, indicating movement of 4 to 8 in.

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[1] Metcalf, R. L., "DDT Substitutes," Crit. Rev. Environ. Contr., 3(1):25-59 (Ref: 110) (1972).

[2] von Rümker, R., and F. Horay, Pesticide Manual, Vol. I, Department of State, Agency for International Development (1972).

These volatilization and leaching index numbers for parathion are in excellent agreement with many laboratory and field observations reviewed in the preceding sections of this report.

It thus appears that volatilization and surface runoff adsorbed on solids are the two most important environmental transport mechanisms for parathion. Surface runoff as a solute in water is likely to occur only very soon after application, before substantial adsorption on soil solids, especially on organic matter, has occurred. Leaching of parathion through soil profiles does not occur readily, and is not likely to result following field applications at rates recommended for insect control.

References

Anderson, B. G., "The Toxicity of Organic Insecticides to Daphnia." In: Transactions of the Second Seminar on Biological Problems in Water Pollution, Cincinnati, Ohio, U.S. Public Health Service, pp. 94-95 (1959). (In: Li and Fleck, 1972.)

Anderson, J. P. E., "Factors Influencing Insecticide Degradation by a Soil Fungus, Mucor Alternans," Diss. Abstr. Int., 32(6):3114B-3115B (1971).

Anon., National Water Quality Laboratory (EPA), unpublished data (24 May 1974).

Applegate, H. G., "Insecticides in the Big Bend National Park," Pest. Monit. J., 4(1):2-7 (1970).

Barker, R. J., "Cholinesterase Reactivators Tested as Antidotes for the Use on Poisoned Honeybees," J. Econ. Entomol., 63(6):1831-1833 (December 1970).

Battelle's Columbus Laboratories, "Effects of Chemicals on Aquatic Life," Water Quality Criteria Data Book, Vol. III, p. B167-B186, Environmental Protection Agency (1971).

Bejer-Petersen, B., R. R. Hermansen, and M. Weihe, "On the Effects of Insecticide Sprayings in Forests on Birds Living in Nest Boxes," Dan. Ornithol. Foren. Iidsskr., 66(1,2):30-50 (1972).

Benke, G. M., K. L. Cheever, F. E. Mirer, and S. D. Murphy, "Comparative Toxicity, Anticholinesterase Action and Metabolism of Methyl Parathion in Sunfish and Mice," Toxicol. Appl. Pharmacol., 28:97-109 (1974).

Bollen, W. B., H. E. Morrison and H. H. Crowell, "Effect of Field Treatments of Insecticides on Numbers of Bacteria, Streptomyces, and Molds in the Soil," J. Econ. Entomol., 47(2):302-306 (1954).

Brodeur, J., and K. P. DuBois, "Comparison of Acute Toxicity of Anticholinesterase Insecticides to Weanling and Adult Male Rats," Proc. Soc. Exp. Biol. Med., 114(2):509-511 (November 1963).

Bucknell, E. S., "Side-Effects of Granulated Insecticides in Canterbury," Proc. N. Z. Weed Pest. Contr. Conf., No. 23:124-126 (1970).

Burkhardt, C. C., and M. L. Fairchild, "Toxicity of Insecticides to House Crickets and Bioassay of Treated Soils in the Laboratory," J. Econ. Entomol., 60(6):1496-1503 (1967a).

Burkhardt, C. C., and M. L. Fairchild, "Bioassay of Field-Treated Soils to Determine Bioactivity and Movement of Insecticides," J. Econ. Entomol., 60(6):L1602-1610 (1967b).

Buttiker, W., "Ecological Effects of Insect Control on Bird Populations," Int. Union Conserv. Nature Natur. Resources, Tech. Meeting, Warsaw 1960, Proc. 8:48-60 (1961). Quoted from: Pimentel (1971).

California Department of Water Resources, San Joaquin Valley Drainage Monitoring Program, 1969 Summary, Sacramento, California (1969). (In: Li and Fleck, 1972.)

California Department of Water Resources, San Joaquin Valley Drainage Monitoring Program, 1970 Summary, Sacramento, California (1970). (In: Li and Fleck, 1972.)

Chambers, J. E., and J. D. Yarbrough, "Parathion and Methyl Parathion Toxicity to Insecticide-Resistant and Susceptible Mosquitofish (Gambusia affinis)," Bull. Environ. Contam. Toxicol., 4:315-320 (1974).

Campbell, W. V., D. A. Mount, and B. S. Heming, "Influence of Organic Matter Content of Soils on Insecticidal Control of the Wireworm," J. Econ. Entomol., 64(1):41-44 (1971).

Chisholm, D., and A. W. MacPhee, "Persistence and Effects of Some Pesticides in Soil," J. Econ. Entomol., 64(4):1010-1013 (August 1972).

Chopra, S. L., and F. C. Khullar, "Degradation of Parathion in Soils," J. Indian So. Soil Sci., 19(1):79-85 (1971).

Cope, O. B., "Contamination of the Freshwater Ecosystem Pesticides," J. Appl. Ecol., 3(Suppl):33-44 (A Special issue on Pesticides in the Environment and Their Effects on Wildlife) (1966). (In: Li Fleck, 1972.)

Coppage, D. L., "Characterization of Fish Brain Acetylcholinesterase with an Automated Ph Stat for Inhibition Studies," Bull. Environ. Contam. Toxicol., 6:304-310 (1971).

Coppage, D. L., "Organophosphate Pesticides: Specific Level of Brain AChE Inhibition Related to Death in Sheepshead Minnows," Trans. Am. Fish. Soc., 101:534-536 (1972).

Coppage, D. L., and E. Matthews, "Short-Term Effects of Organophosphate Pesticides on Cholinesterases of Esturarine Fishes and Pink Shrimp," Bull. Environ. Contam. Toxicol., 11:483-488 (1974).

Cowley, G. T., and E. P. Lichtenstein, "Growth Inhibition of Soil Fungi by Insecticides and Annulment of Inhibition by Yeast Extract or Nitrogenous Nutrients," J. Gen. Microbiol., 62(1):27-34 (1970).

Crockett, A. B., G. B. Wiersma, H. Tai, W. G. Mitchell and P. J. Sand, "National Soils Monitoring Program for Pesticide Residues - FY 1970," U.S. Environmental Protection Agency, Technical Services Division. Unpublished Manuscript (1970).

Croft, B. A., and L. R. Jeppson, "Comparative Studies on Four Strains of Typhlodromus occidentalis, II. Laboratory Toxicity of Ten Compounds Common to Apple Pest Control," J. Econ. Entomol., 63(5): 1528-1531 (October 1970).

Dolphin, R. E., and R. N. Peterson, "Developments in the Research and Control Program of the Clear Lake Gnat Chaoborus astictopus, D & S," Proc. and Papers of Twenty Eighth Ann. Conf., California Mosquito Control Assoc., pp. 90-94 (1960).

Drift, J., "Pesticides and Soil Fauna," Meded, Rijksfac. Landbouwwetensch., Gent, 35(2):707-716 (1970)

Eichelberger, J. W., and J. J. Lichtenberg, "Persistence of Pesticides in River Water," Environ. Sci. Tech., 5(6):541-544 (1971).

Federal Water Pollution Control Administration, "Water Quality Criteria," Report of the National Technical Advisory Committee, p. 37 (1968).

Fukuto, T. R., and J. J. Sims, "Metabolism of Insecticides and Fungicides," Pest. Environ., R. White-Stevens (ed.) Vol. I, Part I, Chapter 2, pp. 147-155, Marcel-Dekker, New York (1971).

Gaufin, A. R., L. D. Jensen, A. V. Nebeker, T. Nelson and R. W. Teel, "The Toxicity of Ten Organic Insecticides to Various Aquatic Invertebrates," Water Sewage Works, 12:276-279 (1965). (In: Li and Flect, 1972.)

Gibson, J. R., J. L. Ludke, and D. E. Ferguson, "Sources of Error in the Use of Fish-Brain Acetylcholinesterase Activity as a Monitor for Pollution," Bull. Environ. Contam. Toxicol., 4:17-23 (1969).

Gibson, J. R., "Comparative Biochemistry of Parathion Metabolism in Three Species of Fishes," Diss. Abstr. Int., 32(4):2365-B (1971).

Graetz, D. A., G. Chesters, T. C. Daniel, L. W. Newland, and G. B. Lee, "Parathion Degradation in Lake Sediments," J. Water Poll. Control Fed., 42(2, Pt. 2):R76-R94 (1970).

Gregory, Jr., W. W., J. K. Reed, and L. E. Priester, Jr., "Accumulation of Parathion and DDT by Some Algae and Protozoa," J. Protozool., 16(1):69-71 (1969).

Greve, P. A., "Toxic Organic Trace Pollutants in Surface Water," Chem. Weekblad., 41(68):11, 13, 15 (1972).

Griffiths, D. C., and N. Walker, "Microbiological Degradation of Parathion," Meded, Rijksfac. Landbouwwetensch., Gent 35(2):805-810 (1970).

Grzenda, A. R., G. J. Lauer, and H. P. Nicholson, "Insecticide Contamination in a Farm Pond, Part II - Biological Effects," Trans. Am. Fish. Soc., 91(2):213-221 (1962).

Gupta, R. S., and K. S. Kushwaha, "Toxicity of Some Insecticides to the Predator, Menochilus sexmaculata," Indian J. Entomol., 32(Pt. 4): 379-381 (1970).

Hamilton, E. W., and R. W. Kieckhefer, "Toxicity of Malathion and Parathion to Predators of the English Grain Aphid," J. Econ. Entomol., 62(5):1190-1192 (October 1969).

Harris, C. R., "Influence of Soil Type on the Insecticidal Activity of Insecticides in Soil," J. Econ. Entomol., 59(5):1221-1225 (1966).

Harris, C. R., and E. P. Lichtenstein, "Factors Affecting the Volatilization of Insecticidal Residues from Soils," J. Econ. Entomol., 54(5):1038-1045 (1961).

Harris, C. R., and W. W. Sans, "Insecticide Residues in Soils on 16 Farms in Southwestern Ontario - 1964, 1966, and 1969," Pest. Monit. J., 5(3):259-267 (December 1971).

Heath, R. G., J. W. Spann, E. F. Hill, J. F. Kreitzer, "Comparative Dietary Toxicities of Pesticides to Birds," Bureau of Sport Fisheries and Wildlife, Special Scientific Report, Wildlife No. 152 (February 1972).

Henderson, C., and Q. H. Pickering, "Toxicity of Organic Phosphorus Insecticides to Fish," Trans. Am. Fish. Soc., 87:39-51 (1958).

Herzel, F., "The Behavior of Several Persistent Insecticides in the Soil," Bundesquesundheitsblatt, 14(3):23-28 (1971).

Holland, H. T., D. L. Coppage, and P. A. Butler, "Use of Fish Brain Acetylcholinesterase to Monitor Pollution by Organophosphorus Pesticides," Bull. Environ. Contam. Toxicol., 2(3):156-162 (1967).

Hubbell, D. H., D. F. Rothwell, W. B. Wheeler, W. B. Tappan, and F. M. Rhoads, "Microbiological Effects and Persistence of Some Pesticide Combinations in Soil," J. Environ. Qual., 2(1):96-99 (1973).

Hudson, R. H., R. K. Tucker, and M. A. Haegele, "Effect of Age on Sensitivity: Acute Oral Toxicity of 14 Pesticides to Mallard Ducks of Several Ages," Toxicol. Appl. Pharmacol., 22:556-561 (1972).

Iwata, Y., W. E. Westlake, and F. A. Gunther, "Persistence of Parathion in Six California Soils Under Laboratory Conditions," Arch. Environ. Contam. Toxicol., 1(1):84-96 (1973).

Jensen, L. D., and A. R. Gaufin, "Effects of Ten Organic Insecticides on Two Species of Stonefly Naiads," Trans. Am. Fish. Soc., 93-34 (1964a). (In: Li and Fleck, 1972.)

Jensen, L. D., and A. R. Gaufin, "Long-Term Effects of Organic Insecticides on Two Species of Stonefly Naiads," Trans. Am. Fish. Soc., 93:357-363 (1964b). (In: Li and Fleck, 1972.)

Johansen, D., "Spray Additives for Insecticidal Selectivity to Injurious vs Beneficial Insects," Environ. Entomol., 1(1):51-54 (February 1972a).

Johansen, C. A., "Toxicity of Field-Weathered Insecticide Residues to Four Kinds of Bees," Environ. Entomol., 1(3):393-394 (June 1972).

Joiner, R. L., and K. P. Baetcke, "Parathion: Persistence on Cotton and Identification of Its Photoalteration Products," J. Agr. Food Chem., 21(3):391-396 (1973).

Kaplan, H. M., and S. S. Glaczenski, "Hematological Effects of Organophosphate Insecticides in the Frog (Rana Pipiens)," Life Sci., 4(7):1213-1219 (1965).

Keith, J. O., M. S. Mulla, "Relative Toxicity of Five Organophosphorus Mosquito Larvicides to Ducks," J. Wildlife Manag., 30(3):553-563 (1966).

Kilgore, W. W., N. Marey, and W. Winterlin, "Parathion in Plant Tissues: New Considerations," In: Degradation of Synthetic Organic Molecules in the Biosphere, National Academy of Sciences, pp. 291-312 (1972).

Kimmerle, G., and D. Lorke, "Toxicity of Insecticidal Organophosphates," Pflanz. Nachr. Bayer., 21:111-142 (1968).

King, P., and P. L. McCarty, "A Chromatographic Model for Predicting Pesticide Migration in Soils," Soil Sci., 106(4):248-261 (1968).

Knutson, H., A. M. Kadoum, T. L. Hopkins, G. F. Swoyer and T. L. Harvey, "Insecticide Usage and Residues in a Newly Developed Great Plains Irrigation District," Pest. Monit. J., 5:17-27 (1971).

Kortus, J., P. Macuch, J. Mayer, K. Durcek, and V. Krcmery, "Uptake of 32-P-Parathion and 32-P Imidan," J. Hyg. Epidemiol. Microbiol. Immunol. (Prague), 15(1):101-3 (1971).

Lahav, M., and S. Sarig, "Sensitivity of Pond Fish to Cotnion (Azinphosmethyl) and Parathion," Bamidgeh, 21(3):67-74 (1970).

Leenheer, J. A., and J. L. Ahlrichs, "A Kinetic and Equilibrium Study of the Adsorption of Carbaryl and Parathion Upon Soil Organic Matter Surfaces," Soil Sci. Am. Proc., 35(5):700-705 (1971).

Lewallen, L. L., and W. H. Wilder, "Toxicity of Certain Organophosphorus and Carbamate Insecticides to Rainbow Trout," Mosquito News, 22 (4): 369-372 (1962).

Li, M., and R. A. Fleck, "The Effects of Agricultural Pesticides in Aquatic Environment, Irrigated Croplands, San Joaquin Valley," Pesticide Studies Series - 6. Environmental Protection Agency, Office of Water Programs, Applied Technology Division, Rural Waste Branch TS-00-72-05, 268 pp. (1972).

Lichtenstein, E. P., "Increase of Persistence and Toxicity of Parathion and Diazinon in Soils with Detergents," J. Econ. Entomol., 59(4):985-993 (1966).

Lichtenstein, E. P., and K. R. Schulz, "The Effects of Moisture and Microorganisms on the Persistence and Metabolism of Some Organophosphorus Insecticides in Soils, with Special Emphasis on Parathion," J. Econ. Entomol., 57:618-627 (1964).

Lichtenstein, E. P., and K. R. Schulz, "Volatilization of Insecticides from Various Substrates," Agr. Food Chem., 18(5):814-818 (1970).

Lichtenstein, E. P., K. R. Schulz, R. F. Skrentny, and Y. Tsukano, "Toxicity and Fate of Insecticide Residues in Water," Arch. Environ. Health, 12:199-212 (1966).

Lichtenstein, E. P., T. W. Fuhremann, K. R. Schulz, and R. F. Skrentny, "Effect of Detergents and Inorganic Salts in Water on the Persistence and Movement of Insecticides in Soils," J. Econ. Entomol., 60(6):1714-1721 (1967).

Lingappa, S. S., K. J. Starks, and R. D. Eikenbary, "Insecticidal Effect on Lysiphlebus testaceipes, a Parasite of the Greenbug, at Three Development Stages," Environ. Entomol., 1(4):520-521 (August 1972).

Lowe, J. I., P. D. Wilson, and R. B. Davison, "Laboratory Bioassays," Progress Report for Fiscal Year 1969, Bureau of Commercial Fisheries Center for Estuarine and Menhaden Research, Pesticides Field Station Gulf Breeze, Florida, U.S. Dept. Interior, Circular 335, pp. 20-28 (1970).

Lowe, J. I., P. D. Wilson, and A. J. Wilson, "Chronic Exposure of Oysters to DDT, Toxaphene and Parathion," Proc. Nat. Shellfish Assoc., 61:71-79 (1971).

Mackiewicz, M., K. H. Deubert, H. B. Gunner, and B. M. Zuckerman, "Study of Parathion Biodegradation Using Gnotobiotic Techniques,", J. Agr. Food Chem., 17(1):129-130 (January/Febraury 1969).

Manescu, S., "The Value of the Manometric Procedure in Studying the Chemical Micropollutant Action Potential on the Self-Purifying Water Flora," Igiena, 20(5):275-286 (1971).

Matida, Y., and N. Kawasaki, "Study on the Toxicity of Agricultural Control Chemicals in Relation to Freshwater Fisheries Management," No. 2 Toxicity of Agricultural Insecticides to *Daphnia carinata*, King. *Bull. Freshwater Fish. Res. Lab.*, Tokyo, 8:1-6 (1968). (In: Li and Fleck, 1972.)

Matsumura, F., and G. M. Boush, "Metabolism of Insecticides by Microorganisms," *Soil Biochem.*, 320-336, Marcel Dekker, New York (1971).

McFarland, L. Z., and P. B. Lacy, "Acute Anticholinesterase Toxicity in Ducks and Japanese Quail," *Toxicol. Appl. Pharmacol.*, 12:105-114 (1968).

Metcalf, R. L., "DDT Substitutes," *Crit. Rev. Environ. Contr.*, 3(1): 25-59 (Ref: 110) (1972).

Mick, D. L., and P. A. Dahm, "Metabolism of Parathion by Two Species of Rhizobium," *J. Econ. Entomol.*, 63(4):1155-1159 (August 1970).

Miller, C. W., B. M. Zuckerman, and A. J. Charig, "Water Translocation of Diazinon-C^{14} and Parathion-S^{35} off a Model Cranberry Bog and Subsequent Occurrence in Fish and Mussels," *Trans. Am. Fish. Soc.*, 95:345-349 (1966).

Moffitt, H. R., E. W. Anthon, and L. O. Smith, "Toxicity of Several Commonly Used Orchard Pesticides to Adult *Hippodamia convergens*," *Environ. Entomol.*, 1(1):20-23 (February 1972).

Mol, J. C. M., D. L. Harrison, and R. H. Talfer, "Parathion: Toxicity to Sheep and Persistence on Pasture and in Soil," *N. Z. J. Agr. Res.*, 15(2):306-320 (1972).

Moore, R. B., "Effects of Pesticides on Growth and Survival of *Euglena gracilis Z.*, *Bull. Environ. Contam. Toxicol.*, 5(3):226-230 (1970).

Mulla, M. S., "Vector Control Technology and Its Relationship to the Environment and Wildlife," *J. Appl. Ecol.*, **3** (Supplement on Pesticides in the Environment and Their Effects on Wildlife): 21-28 (1966). Quoted from: Pimentel (1971).

Mulla, M. S., J. St. Amant, and L. D. Anderson, "Evaluation of Organic Pesticides for Possible Use as Fish Toxicants," *Prog. Fish-Cult.*, 29(1):36-42 (1967).

Mulla, M. S., and L. W. Isaak, "Field Studies on the Toxicity of Insecticides to the Mosquitofish, Gambusia affinis," J. Econ. Entomol., 54(6):1237-1242 (1961).

Mulla, M. S., L. W. Isaak, and H. Axelrod, "Field Studies on the Effects of Insecticides on Some Aquatic Wilflife Species," J. Econ. Entomol., 56(2):184-188 (1963).

Muncy, R. J., and A. D. Oliver, Jr., "Toxicity of Ten Insecticides to the Red Crawfish, Procambarus clarki (Girard)," Trans. Am. Fish. Soc., 92:428-431 (1963). (In: Li and Fleck, 1972.)

Murphy, S. D., R. R. Lauwerys, and K. L. Cheever, "Comparative Anticholinesterase Action of Organophosphorus Insecticides in Vertebrates," Toxicol. Appl. Pharmacol., 12:22-35 (1968).

Nagvi, S. M., and D. E. Ferguson, "Levels of Insecticide Resistance in Freswater Shrimp, Palaemonetes kadiakensis," Trans. Am. Fish Soc., 99:696-699 (1970). (In: Li and Fleck, 1972.)

Nagvi, S. M., and D. E. Ferguson, "Levels of Insecticides Resistance in Freshwater Shrimp, Palaemonetas kadiakensis," Trans. Am. Fish. Soc., 99:697 (1970).

Nicholson, H. P., H. J. Webb, G. J. Lauer, R. E. O'Brien, A. R. Grzenda, and D. W. Shanklin, "Insecticide Contamination in a Farm Pond. Part I - Origin and Duration," Trans. Am. Fish. Soc., 91(2):213-221 (1962).

Nishiuchi, Y., and Y. Hashimoto, "Toxicity of Pesticides to Some Freshwater Organisms," Rev. Plant Protec. Res., 2:137-139 (1969).

Paris, D. F., and D. L. Lewis, "Chemical and Microbial Degradation of Ten Selected Pesticides in Aquatic Systems," Res. Rev., 45: 95-124 (1973).

Pickering, Q. H., C. Henderson, and A. E. Lemke, "The Toxicity of Organic Phosphorus Insecticides to Different Species of Warmwater Fishes," Trans. Am. Fish. Soc., 91(2):175-184 (1962).

Pimentel, D., "Ecological Effects of Pesticides on Nontarget Species," Executive Office of the President, Office of Science and Technology, U.S. Government Printing Office, Washington, D.C. (1971).

Poorman, A. E., "Effects of Pesticides on Euglena gracilis, I. Growth Studies," Bull. Environ. Contam. Toxicol., 10(1):25-28 (1973).

Priester, L. E., "The Accumulation in Metabolism of DDT, Parathion and Endrin by Aquatic Food Chain Organisms," Ph.D. Thesis, Clemson University (1965). (In: Li and Fleck, 1972.)

Quentin, K. E., "Pesticides in Water - Determination, Removal and Limiting Values," Chem. Ing. Tech., 44(20):1172-1176 (1972a).

Quentin, K. E., "Evaluation and Significance of Residue Values from the Viewpoint of the Water Biocenosis and Drinking Water Quality," Schriftenr. Ver. Wasser-Boden-Lufthyg., 34:19-28 (1972b).

Robel, R. J., C. D. Stalling, M. E. Westfahl, and A. M. Kadoum, "Effects of Insecticides on Populations of Rodents in Kansas - 1965 to 1969," Pest. Monit. J., 6(2):115-121 (September 1972).

Rudd, R. L., and R. E. Genelly, Pesticides: Their Use and Toxicity in Relation to Wildlife, State of California Department of Fish and Game, Game Bull. No. 7, pp. 108-113 (1956).

Sacher, R. M., G. F. Ludvik, and J. M. Deming, "Bioactivity and Persistence of Some Parathion Formulations in Soil," J. Econ. Entomol., 65(2):329-332 (April 1972).

Saltzman, S., L. Kliger, and B. Yaron, "Adsorption-Desorption of Parathion as Affected by Soil Organic Matter," J. Agr. Food Chem., 20(6):1224-1226 (1972).

Saltzman, S., B. Yaron, "Parathion Adsorption from Aqueous Solutions as Influenced by Soil Components," In: Fate of Pesticides in Environment, Gordon and Breach, London, pp. 87-100 (1972).

Sanders, H. O., "Toxicity of Pesticides to the Crustacean Gammarus lacustris," Technical Papers No. 25, pp. 18, United States Department of the Interior Fish and Wildlife Service (1969).

Sanders, H. O., "Pesticide Toxicities to Tadpoles of the Western Chorus Frog Pseudacris triseriata and Fowler's Toad Bufo woodhousii fowleri, Copeia, 2:246-251 (1970).

Sanders, H. O., and O. B. Cope, "Toxicities of Several Pesticides to Two Species of Cladocerans," Trans. Am. Fish. Soc., 95(2):165-169 (1966).

Sanders, H. O., and O. B. Cope, "The Relative Toxicities of Several Pesticides to Naiads of Three Species of Stoneflies," Limnology and Oceanography, 13(1):112-117 (1968).

Sandi, E., "Reduction of Parathion Induced by Light," Nature, 181: 499 (1958).

Satpathy, J. M., G. K. Padhi, and D. N. Dutta, "Toxicity of Eight Insecticides to the Coccinellid Predator *Chilomenes sexmaculata*," Indian J. Entomol., 30(1):130-132 (1968).

Schaefer, C. H., E. Dupras, Jr., "The Effects of Water Quality, Temperature and Light on the Stability of Organophosphorus Larvicides Used for Mosquito Control," Proc. Papers Ann. Conf. California Mosquito Control Assoc. 37:67-75 (1969).

Schafer, E. W., "The Acute Oral Toxicity of 369 Pesticidal, Pharmaceutical and Other Chemicals to Wild Birds," Toxicol. Appl. Pharmacol., 21:315-330 (1972).

Schafer, E. W., and R. B. Brunton, N. F. Lockyer, and J. W. DeGrazio, "Comparative Toxicity of 17 Pesticides to the *Quelea*, House Sparrow, and Red-Winged Blackbird," Toxicol. Appl. Pharmacol., 26:154-157 (1973).

Sethunathan, N., "Degradation of Parathion in Flooded Acid Soils," J. Agr. Food Chem., 21(4):602-604 (1973).

Sethunathan, N., and T. Yoshida, "Conversion of Parathion to Paranitrophenol by Diazinon Degrading Bacterium," Proc. Inst. Environ. Sci. Annu. Tech. Meet., 18:255-257 (1972).

Sethunathan N., and T. Yoshida, "Parathion Degradation in Submerged Rice Soils in the Philippines," J. Agr. Food Chem., 21(3):504-506 (1973).

Sreenivasan, A., and G. K. Swaminathan, "Toxicity of Six Organophosphorus Insecticides to Fish," Curr. Sci., India, 36:397-398 (August 1967).

Stanley, C. W., J. E. Barney II, M. R. Helton, and A. R. Yobs, "Measurement of Atmospheric Levels of Pesticides," Environ. Sci. Tech., 5(5):430-435 (1971).

Stephen, W. P., "The Effects of Chronic Sublethal Doses of Pesticides on Behavior and Longevity in Arthropods," Oregon State Univ. Environ. Health Sci. Cent. Annu. Prog. Rep., 161-167 (1972).

Stevens, L. J., C. W. Collier, and D. W. Woodham, "Monitoring Pesticides in Soils from Areas of Regular, Limited, and No Pesticide Use," Pest. Monit. J., 4(3):145-164 (December 1970).

Stewart, D. K. R., D. Chisholm, and M. T. H. Ragab, "Long-Term Persistence of Parathion in Soil," Nature, 229:47 (1971).

Swoboda, A. R., and G. W. Thomas, "Movement of Parathion in Soil Columns," J. Agr. Food Chem., 16(6):923-927 (November/December 1968).

Teotia, T. P. S., and G. C. Tiwari, "Toxicity of Some Important Insecticides to the Coccinellid Predator, Coccinella septempunctata," Labdev, Part B, 10(1):17-18 (1972).

Tessari, J. D., and D. L. Spencer, "Air Sampling for Pesticides in the Human Environment," J. Assoc. Offic. Anal. Chem., 54(6):1376-1382 (1971)

Tucker, R. K., and D. G. Crabtree, "Handbook of Toxicity of Pesticides to Wildlife," U.S. Department of the Interior, Fish and Wildlife Service, Denver Wildlife Research Center, Resource Publication No. 84, p. 82 (1970).

Tucker, R. K., and M. A. Haegele, "Comparative Acute Oral Toxicity of Pesticides to Six Species of Birds," Toxicol. Appl. Pharmacol., 20:57-65 (1971).

USDI, "Wildlife Research; Problems, Programs and Progress, Pesticide-Wildlife Relations," Fish Wildlife Service, Bur. Sport Fish Wildl. Circ. 43, 117 pp. (1966). Quoted from: Pimentel (1971).

Voerman, S., and A. F. H. Besemer, "Residues of Dieldrin, Lindane, DDT, and Parathion in a Light Sandy Soil After Repeated Application Throughout a Period of 15 Years," J. Agr. Food Chem., 18(4):717-719 (1970).

von Rumker, R., and F. Horay, Pesticide Manual, Vol. I. Department of State, Agency for International Development (1972).

WADC, Wright-Patterson Air Force Base, Ohio, Air Force Systems Command, "The Handbook of Toxicology, Vol. III, "Insecticides" WADC Tech. Report 55-16 (1959).

Ware, G. W., and C. C. Roan, "Interaction of Pesticides with Aquatic Microorganisms and Plankton," Residue Rev., 33:15-45 (1971).

Weidhaas, D. E., M. C. Bowman, and C. H. Schmidt, "Loss of Parathion and DDT to Soil from Aqueous Dispersions and Vermiculite Granules," J. Econ. Entomol., 54(1):175-177 (1961).

Wiersma, G. B., W. G. Mitchell, and C. L. Stanford, "Pesticide Residues in Onions and Soil - 1969," Pest. Monit. J., 5(4):345-347 (March 1972a).

Wiersma, G. B., H. Tai, and P. F. Sand, "Pesticide Residue Levels in Soils, FY 1969 - National Soils Monitoring Program," Pest. Monit. J., 6(3):194-228 (December 1972b).

Wolfe, H. R., and W. F. Durham, "Spillage of Pesticides and Residues in Soil," Washington State Hort. Assoc. Proc., 62:91-92 (1966).

Wolfe, H. R., D. C. Staiff, J. F. Armstrong, and S. W. Comer, "Persistence of Parathion in Soil," Bull. Environ. Contam. Toxicol., 10(1):1-9 (1973).

Wood, E. M., "The Pathology of Pesticide Toxicity in Fish," Unpublished, USDI, Columbia, Mo

Yaron, B., and S. Saltzman, "Influence of Water and Temperature on Adsorption of Parathion by Soils," Soil Sci. Soc. Am. Proc., 36(4): 583-586 (1972).

Zuckerman, B. M., K. Deubert, M. Mackiewicz, and H. Gunner, "Studies on the Biodegradation of Parathion," Plant Soil, 33(2):273-281 (1970).

SUBPART II. D. PRODUCTION AND USE

CONTENTS

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This subsection contains data on the production and uses of parathion; it is organized according to three subject areas: registered uses, production and domestic supply, and use patterns.

Registered Uses of Parathion

Federally Registered Uses - Parathion has a very broad spectrum of effectiveness against insects and mites. It is registered and recommended in the United States for use on a large number of crops, including fruit, nut, vegetable and field crops. Tolerances for parathion residues have been established on about 100 raw agricultural commodities.

All registered uses of parathion by crops, target pests, dosage rates, formulations, type of use, established tolerances, and use, timing, and preharvest interval limitations are summarized in the "EPA Compendium of Registered Pesticides," pages III-P-2.1 through 2.164. This section on parathion, comprising 164 pages, makes up more than 15% of all listings of all registered insecticides, acaricides, molluscidies and antifouling compounds in Volume III of the EPA "Compendium of Registered Pesticides." This fact illustrates the large number and variety of pest control uses for which parathion formulations are currently registered in the U.S.

The registered uses of parathion are detailed in this section by a set of four tables:

Table 29: Parathion - Summary of registered Uses by Crops, Application Rates, and Rate and Time Registration.

Table 30: Pest Insects and Mites against which parathion is recommended, in alphabetical order by common name.

Tables 31 and 32: Registered uses of one of the common formulations of parathion, i.e., emulsifiable liquid containing 4 lb of active ingredient per gallon, by crops; insects and other pests controlled on each crop; recommended dosage rates; and general and specific directions for, and limitations of use.

In these tables, rates of application are given in terms of pints or quarts of formulated product per acre or per volume of spray. These rates are readily convertible into active ingredient units.

TABLE 29. PARATHION - SUMMARY OF REGISTERED USES BY CROPS, APPLICATION RATES, AND RATE AND TIME RESTRICTIONS.

Target crops	Rate per application (lb. AI)	Maxium permissible AI rate. (lb./acre)	Minimum time, last treatment to harvest. (days)
Agricultural crop			
Alfalfa	0.25-0.8/A		15days thru 0.8 lb/A
Almonds 1/	0.25-0.5/100gal. 1.0-2.6/A	2.6	thru 2.6 lb/A
Apples	0.12-0.5/100gal. 0.3-3.0/A	6.0	14days thru 6.0 lb/A
Apricots	0.15-0.5/100gal. 1.0-3.0/A	3.6	14days thru 3.6 lb/A
Artichokes	0.35-0.1/A		7days thru 1.0 lb/A
Avacados	0.18-0.37/100gal.	2.5	21days thru 2.5 lb/A
Barley	0.25-0.75/A		15days thru 0.85 lb/A
Beans	0.25-0.8/A		7days thru 0.5 lb/A 15days> 0.5 thru 0.8 lb/A
(soil appl.)	5.0-6.0/A		
Beets	0.15-0.6/A		15days thru 0.6 lb/A 21days thru 0.6 lb/A 2/
(soil appl.)	2.0-6.0/A		
Blackberries	0.11-1.0/A		15days thru 1.0 lb/A
(soil appl.)	1.0/A		
Black-eyed peas	0.25-0.5/A		15days thru 0.5 lb/A
(soil appl.)	3.0-4.0/A		
Blueberries	0.25-0.5/A		14days thru 0.5 lb/A
Boysenberries	0.15-1.0/A		15days thru 1.0 lb/A
(soil appl.)	1.0/A		
Broccoli	0.15-1.0/A		7days thru 0.5 lb/A 21days> 0.5 thru 1.0 lb/A
(soil appl.)	3.0-6.0/A		
Brussels sprouts	0.15-1.0/A		7days thru 0.5 lb/A 21days 0.5 thru 1.0 lb/A
(soil appl.)	3.0-6.0/A		
Cabbage	0.15-1.0/A		7days thru 0.25 lb/A 10days> 0.25 thru 0.5 lb/A 21days> 0.5 thru 1.0 lb/A
(soil appl.)	3.0-6.0/A		
Cantaloupes	0.15-0.5/A		7days thru 0.5 lb/A
(soil appl.)	3.0-6.0/A		
Carrots	0.2-0.6/A		15days thru 0.6 lb/A
(soil appl.)	2.0-5.0/A		
Cauliflower	0.15-1.0/A		7days thru 0.5 lb/A 21days 0.5 thru 1.0 lb/A
(soil appl.)	3.0-6.0/A		

Source: U.S. Environmental Protection Agency, EPA Compendium of Registered Pesticides, Vol. III, (1973).

Table 29. (Continued)

Celery	0.15-0.75/A		15days thru 0.25 lb/A
			21days > 0.25 thru 0.5 lb/A
			30days > 0.5 thru 0.75 lb/A
(soil appl.)	3.0-5.0/A		
Cherries	0.11-0.5/100gal.	2.0	14days thru 2.0 lb/A
	0.4-1.4/A		
Citrus	0.0625-2.0/100gal.	4.0	14days thru 4.0 lb/A
	1.0-10.0/A	10.0	30days > 4.0 thru 10.0 lb/A
Clover	0.12-0.75/A		15days thru 0.8 lb/A
Collards	0.15-0.5/A		7days thru 0.25 lb/A
			10days > 0.25 thru 0.5 lb/A
(soil appl.)	3.0-4.2/A		
Corn	0.16-0.75/A		12days thru 1.0 lb/A
	0.015/1,000sq.ft.		
(soil appl.)	1.0-6.0/A		
Corn (sweet)	2.0-5.0/A		
(soil appl.)			
Cotton	0.1-1.0/A		7days thru 1.0 lb/A
Cow peas	0.45/A		15days thru 0.45 lb/A
Cranberries	0.5-1.0/100gal.	1.0	15days thru 0.8 lb/A
	0.5-1.0/A		30days > 0.8 thru 1.0 lb/A
Cucumbers	0.1/5x10^{4}cu.ft.		15days thru 0.1/5x10^4 cu.ft.[9]
	0.15-0.5/A		15days thru 0.5 lb/A
(soil appl.)	3.0-4.2/A		
Currants	0.11-0.8/A		30days thru 0.8 lb/A
Dewberries	0.2-0.5/A		15days thru 0.5 lb/A
Eggplant	0.2-0.6/A		15days thru 0.6 lb/A
(soil appl.)	3.0-5.0/A		
Endive (Escarole)	0.1/5x100 cu.ft.		21days > 0.11 thru 5x10 cu.ft.[9]
	0.35-0.5/A		21days thru 0.5 lb/A
(soil appl.)	3.0-4.2/A		
Figs	0.25-0.5/100gal.	2.5	30days thru 2.5 lb/A
Filberts[1]	0.25/100gal.	2.5	thru 2.5 lb/A
	0.4/A		
Garlic	0.25-0.5/A		15days thru 0.5 lb/A
Gooseberries	0.11-0.8/A		15days thru 0.8 lb/A
Grapefruit	0.0625-2.0/100gal.	4.0	14days thru 4.0 lb/A
	1.0-10.0/A	10.0	30days > 4.0 thru 10.0 lb/A
Grapes	0.25-1.0/100gal.	2.5	14days thru 1.5 lb/A
	0.25-2.5/A		1.5 thru 2.5 lb/A[3]
Grass (forage)	0.25-0.8/A		15days thru 0.8 lb/A
Hops	0.25-0.8/A		15days thru 0.8 lb/A
Kale	0.15-0.5/A		7days thru 0.25 lb/A
			10days 0.25 thru 0.5 lb/A
(soil appl.)	3.0-5.0/A		
Kohlrabi	0.2-1.0/A		7days thru 0.8 lb/A
			21days > 0.8 thru 1.0 lb/A
Kumquats	0.0625-2.0/100gal.	4.0	14days thru 4.0 lb/A
	1.0-10.0/A	10.0	30days > 4.0 thru 10.0 lb/A
Lemons	0.0625-2.0/100gal.	4.0	14days thru 4.0 lb/A
	1.0-10.0/A	10.0	30days > 4.0 lb. thru 10.0 lb/A

Table 29. (Continued)

Lettuce	0.1/$5x10^4$ cu.ft.		21days 0.1 lb. thru $5x10^4$ cu.ft.[9]
	0.25-0.5/A		7days thru 0.5 lb/A (head)
			14days thru 0.25 lb/A (bibb & head)
			21days > 0.25-0.5 lb/A (bibb & head)
(soil appl.)	3.0-6.0/A		
Limes	0.0625-2.0/100g	10.0	14days thru 4.0 lb/A
Loganberries	0.25-1.0/A		15days thru 1.0 lb/A
Mangoes	0.5/100gal.	3.0	21days thru 3.0 lb/A
Melons	0.15-0.5/A		7days thru 0.5 lb/A
(soil appl.)	4.0-6.0/A		
Mustard greens	0.15-0.5/A		7days thru 0.25 lb/A
			10days > 0.25 thru 0.5 lb/A
Nectarines	0.18-0.5/100gal.	5.0	14days thru 4.0 lb/A
	0.6-2.0/A		21days thru 2.5 lb/A[4]
Oats	0.25-0.75/A		15days thru 0.75 lb/A
Okra	0.25-0.75/A		21days thru 0.75 lb/A
Olives	0.25-0.62/100gal.	5.0	
Onions	0.2-0.8/A		15days thru 0.8 lb/A[5]
(soil appl.)	0.38-0.6/A		
Oranges	0.0625-2.0/100gal.	4.0	14days thru 4.0 lb/A
	1.0-10.0/A	10.0	30days > 4.0 thru 10.0 lb/A
Pastures see Grass (forage)			
(soil appl.)	0.5-1.0/A		7days thru 0.1 lb/A
Pastures (irrigated)	0.1/A		7days thru 0.116/A
Peaches	0.18-0.5/100gal.	5.0	14days thru 4.0 lb/A
	0.6-2.0		21days thru 2.5 lb/A[4]
Peanuts	0.2-0.5/A		15days thru 0.5 lb/A
(soil appl.)	2.0-3.0/A		
Pears	0.15-0.38/100gal.	3.6	14days thru 3.6 lb/A
	0.4-1.0/A		
Peas	0.25-0.5/A		10days thru 0.5 lb/A (peas)
			15days thru 0.5 lb/A (forage)
(soil appl.)	3.0-6.0/A		
Pecans	0.25-0.38/100gal.	5.4	15days thru 5.4 lb/A
	0.6-0.7/A		
Peppers	0.15-0.8/A		15days thru 0.8 lb/A
(soil appl.)	5.0-6.0/A		
Pineapples	0.45-0.75/A		7days thru 0.75 lb/A
(preplant dip)	0.15/100gal.		
Plums/Prunes	0.25-0.5/100gal.	4.0	14days thru 4.0 lb/A
	1.0-1.5/A		
Potatoes	0.25-1.0/A		5days thru 1.0 lb/A
(soil appl.)	2.0-6.0/A		
Pumpkins	0.2-0.5/A		10days thru 0.5 lb/A
Quince	0.3-0.38/100gal.		14days thru 3.6 lb/A

Table 29. (Continued)

Radishes	0.1/$5x10^4$cu.ft.		15days thru 0.1/$5x10^4$ cu.ft.9/
Raspberries	0.2-1.0/A		15days thru 1.0 lb/A
Rice	0.094-0.1/A		1day thru 0.1 lb/A
Rutabagas	0.15-0.75/A		7days thru 0.75 lb/
(soil appl.)	3.0-5.0/A		
Safflower	0.5/A		thru 0.5 lb/A6/
Sorghum	0.25-1.01 lb/A		12days thru 1.0 lb/A
Soybeans	0.2-0.8/A		20days thru 0.8 lb/A
(soil appl.)	3.0-4.2/A		
Spinach	0.2-0.5/A		14days thru 0.5 lb/A
(soil appl.)	4.0-6.0/A		
Squash	0.15-0.5/A		15days thru 0.5 lb/A
Strawberries	0.2-0.8/A		14days thru 0.8 lb/A
	1.0/A7/		
(soil appl.)	0.4-5.0/A		
Sugar beets	0.3-0.8/A		15days thru 0.8 lb/A
(soil appl.)	4.0-5.0/A		
Sugarcane	2.0-6.0/A		
(soil appl.)			
Sweet potatoes	0.5-1.0/A		15days thru 11.0 lb/A
(soil appl.)	2.0-4.0/A		
Swiss chard	0.2-0.5/A		21days thru 0.5 lb/A
Tangelos/ Tangerines	0.0625-2.0/100gal.	4.0	14days thru 4.0 lb/A
	1.0-10/A	10.0	3days>4.0 thru 10.0 lb/A
Tobacco	0.01-0.05/100sq.yd.8/		5days thru 0.5 lb/A (by priming)
	0.15-0.5/A		15days thru 0.5 lb/A (by cutting)
(soil treat)	2.0-6.0/A		
Tomatoes	0.1/5x10 sq.ft.		10days thru 0.1/10^4 sq.ft.9/
	0.25-1.0/A		10days thru 1.0 lb/A
(soil appl.)	3.0-6.0/A		
Turnips	0.25-0.5/A		7days thru 0.25 lb/A
			10days>0.25 thru 0.5 lb/A
(soil appl.)	3.0-5.0/A		
Vetch	0.25-0.8/A		15days thru 0.8 lb/A
Walnuts10/	0.075-0.5/100gal	5.0	15days thru 0.8 lb/A
	0.3-0.8/A		
Watermelons	0.2-0.5/A		7days thru 0.5 lb/A
(soil appl.)	3.0-4.0/A		
Wheat	0.25-0.8/A		15days thru 1.0 lb/A

Ornamentals

Gladiolus (soil appl.)	5.0/A
Flowering plants	0.15-0.75/A
(soil appl.)	2.0-5.0/A

Table 29. (Continued)

Woody Shrubs, Trees and Vines		
Christmas trees	0.25/100gal.	
Shrubs/Trees/Vines	0.15-0.75/A	
(soil appl.)	2.0-5.0/A	
Greenhouse Plants		
Chrysanthemums	0.1/5x10^4 sq.ft.	For commercial use only.
Lilies	0.1/5x10^4 sq.ft.	For commercial use only.
Greenhouse ornamentals	0.1/5x10^4 sq.ft.	For commercial use only.
Aquatic Areas		
Standing water	0.04/A	See restrictions listed in Compendium.

Notes:

1. Do not apply after hulls begin to open thru 2.6 lb/A.
2. 30 day spray/harvest interval if tops are to be used as food or feed.
3. Apply dosages > 1.5 thru 2.5 lb./A before fruit is the size of buckshot or after harvest.
4. California: Do not apply 5.0lb/A between Jan. 1 and harvest.
5. Do not apply later than Aug. 1 through 5.0 lb/A.
6. Do not apply after flowering through 5.0 lb/A.
7. For post-harvest only.
8. Seed bed treatment.
9. For commercial greenhouse use only.
10. Do not apply after husks open through 5.0 lb/A.

TABLE 30. PEST INSECTS AND MITES AGAINST WHICH PARATHION IS RECOMMENDED (IN ALPHABETICAL ORDER BY COMMON NAMES)

Alfalfa caterpillar	*Colias eurytheme*
Alfalfa looper	*Autographa californica*
Alfalfa seed chalcid	*Bruchophagus roddi*
Alfalfa weevil	*Hypera postica*
American cockroach	*Periplaneta americana*
American plum borer	*Euzophera semifuneralis*
Ants	Family Formicidae
*Aphids	Family Aphididae
Apple flea weevil	*Rhynchaenus pallicornis*
Apple lace bug	*Corythucha caelata*
Apple maggot	*Rhagoletis pomonella*
Apple redbug	*Lygidea mendax*
Armyworm	*Pseudaletia unipuncta*
Artichoke plume moth	*Platyptilia carduidactyla*
Asiatic garden beetle	*Maladera castanea*
Avocado lace bug	*Pseudacysta perseae*
Avocado leafhopper	*Idona minuenda*
Azalea leafminer	*Gracillaria azaleelia*
Bagworm	*Thyridopteryx ephemeraeformis*
Banded cucumber beetle	*Diabrotica balteata*
Beet armyworm	*Spodoptera exigua*
Bean leaf beetle	*Erynephala puncticollis*
Beet leafhopper	*Circulifer tenellus*
Bean leafroller	*Urbanus proteus*
Black grass bugs	Family Miridae
Black vine weevil	*Otiorhynchus sulcatus*
Blackheaded fireworm	*Rhopobota naevana*
Black vine weevil	*Otiorhynchus sulcatus*
Blister beetles	Family Meloidae
Blossom anomala	*Anomala undulata*
Blossom weevils	*Anthonomus* spp.
Blueberry maggot	*Rhagoletis mendax*
Boll weevil	*Anthonomus grandis*
Boll worm	*Heliothis zea*
Cabbage looper	*Trichoplusia ni*
Cankerworms	Family Geometridae
Carrot rust fly	*Psila rosae*
*Casebearers	Family Coleophoridae
Catfacing insects	Order Hemiptera/Heteroptera
Celery leaftier	*Udea rubigalis*
Cherry fruitworm	*Grapholitha packardi*
Cherry maggot	*Rhagoletis cingulata*
Chinch bug	*Plissus leucopterus*
Citrus root weevil	*Pachnaeus litus*
Climbing snails	Class Gastropoda
Clover head weevil	*Hypera meles*
Clover leaf weevil	*Hypera punctata*
Codling moth	*Laspeyresia pomonella*
Colorado Potato beetle	*Leptinotarsa decemlineata*
Consperse stinkbug	*Euschistus conspersus*

Source: List of pest insects and mites from: U.S. Environmental Protection Agency, *EPA Compendium of Registered Pesticides*, Vol. III (1973); scientific names from: Supplement 13 to *EPA Compendium*, Vol. III.

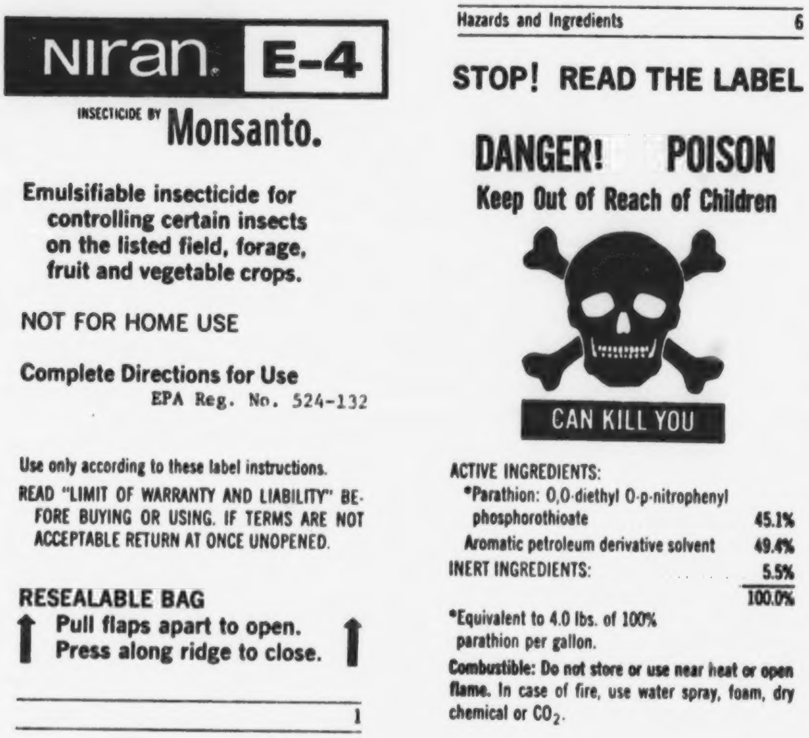
Table 30. (Continued)

Corn earworm	*Heliothis zea*
Corn rootworms	*Diabrotica* spp.
Corn silk fly	*Euxesta stigmatias*
Cotton leafperforator	*Bucculatrix thurberiella*
Cotton leafworm	*Alabama argillacea*
Cranberry fruitworm	*Acrobasis vaccinii*
Cranberry tipworm	*Dasineura oxycoccana*
Crane flies	Family Tipulidae
Crickets	Family Gryllidae
Cucumber beetles	Family Chrysomelidae
Current borer	*Synanthedon tipuliformis*
*Cutworms	Family Noctuidae
Darkling beetles	Family Tenebrionidae
Diamondback moth	*Plutella xylostella*
Earwigs	Family Dermaptera
European apple sawfly	*Hoplocampa testudinea*
European Corn borer	*Ostrinia nubilalis*
Eyespotted bud moth	*Spilonota ocellana*
Fall armyworm	*Spodoptera frugiperda*
Fall webworm	*Hyphantria cunea*
False celery leaftier	*Udea profundalis*
False chinch bug	*Nysius ericae*
Flea beetles	Family Chrysomelidae
Fruittree leafroller	*Archips argyrospilus*
Fuller rose beetle	*Pantomorus cervinus*
Grape berry moth	*Paralobesia viteana*
Grape bud beetle	*Glyptoscelis squamulata*
Grape leaffolder	*Desmia funeralis*
Grasshoppers	Family Acrididae
Green cloverworm	*Plathypena scabra*
Green fruitworm	*Lithophane antennata*
Green June beetle	*Cotinis nitida*
Greenhouse leaftier	*Udea rubigalis*
Harlequin bug	*Murgantia histionica*
Hoplia beetles	*Hoplia* spp.
Hornworms	Family Sphingidae
Imported cabbageworm	*Pieris rapae*
Japanese beetle	*Popillia japonica*
Juniper webworm	*Dichomeris marginella*
Katydids	Family Tettigoniidae
Lace bugs	Family Tingidae
Leafhoppers	Family Cicadellidae
Leaffooted bug	*Leptoglossus phyllopus*
*Leafminers	Class Insecta
*Leafrollers	Class Insecta
*Leaftiers	Order Lepidoptera
Lesser appleworm	*Grapholitha prunivora*
Lesser cornstalk borer	*Elasmopalpus lignosellus*
Lesser peachtree borer	*Synanthedon pictipes*
Limabean pod borer	*Etiella zinckenella*
Little fire ant	*Wasmannia auropunctata*

Table 30. (Continued)

Lygus bug	*Lygus* spp.
Mealybugs	Family Pseudococcidae
Mellonworm	*Diaphania hyalinata*
Midges	Family Chironomidae
*Mites	Class Acarina
Mosquitoes	Family Culicidae
Obsure root weevil	*Sciophithes obscurus*
Omnivorous leaf roller	*Platynota stultana*
Onion maggot	*Hylemya antiqua*
Orangedog	*Papilio cresphontes*
Orange worm	Class insecta
Orange tortrix	*Argyrotaenia citrana*
Oriental fruit moth	*Grapholitha molesta*
Pameras	*Pachybrachius bilobata*
Pandemis moth	Family Tortricidae
Peach bark beetle	*Philoeotribus liminaris*
Peach twig borer	*Anarsia lineatella*
Peachtree borer	*Sanninoidea exitiosa*
Pea moth	*Laspeyresia nigricana*
Pea weevil	*Bruchus pisorum*
Pearslug	*Caliroa cerasi*
Pear psylla	*Psylla pyricola*
Pecan leaf casebearer	*Acrobasis juglandis*
Pecan nut casebearer	*Acrobasis caryae*
Pepper maggot	*Zonosemata electa*
Pickleworm	*Diaphania nitidalis*
Pink scavenger caterpillar	*Sathrobrota rileyi*
Plant bugs	Family Miridae
Plum curculio	*Conotrachelus nenuphar*
Potato psyllid	*Paratioza cockerelli*
Potato tuberworm, Splitowrm	*Phthorimaea operculella*
Psyllids	Family Psyllidae
Raspberry crown borer	*Bembecia marginata*
Readbanded leafroller	*Argyrotaenia velutinana*
Readnecked cane borer	*Agrilus ruficollis*
Readnecked peanutworm	*Stegasta bosquella*
Rice leafminer	*Hydrellia griseola*
Rindworms	Order Lepidoptera
Rose chafer	*Macrodactylus subspinosus*
Saltmarsh caterpillar	*Estigmene acrea*
Sand wireworm	*Horistonotus uhlerii*
Sapbeetles	Family Nitidulidae
Sawflies	Order Hymenoptera
*Scales	Superfamily Coccoidea
Seedcorn maggot	*Hylemya platura*
Shothole borer	*Scolytus rugulosus*
Sixspotted leafhopper	*Macrosteles fascifrons*
Sorghum midge	*Contarina sorghicola*
Sorghum webworm	*Celama sorghiella*
Sowbugs	Order Isopoda

Table 31. REGISTERED USES OF PARATHION EMULSIFIABLE LIQUID (4 LB ACTIVE INGREDIENT PER GALLON) - CROPS AND OTHER USES, PESTS, DOSAGE RATES AND USE LIMITATIONS[a/]



Niran. E-4

INSECTICIDE BY **Monsanto.**

Emulsifiable insecticide for controlling certain insects on the listed field, forage, fruit and vegetable crops.

NOT FOR HOME USE

Complete Directions for Use

EPA Reg. No. 524-132

Use only according to these label instructions.

READ "LIMIT OF WARRANTY AND LIABILITY" BEFORE BUYING OR USING. IF TERMS ARE NOT ACCEPTABLE RETURN AT ONCE UNOPENED.

RESEALABLE BAG

↑ Pull flaps apart to open. Press along ridge to close. ↑

1

Hazards and Ingredients 6

STOP! READ THE LABEL

DANGER! POISON

Keep Out of Reach of Children

CAN KILL YOU

ACTIVE INGREDIENTS:	
*Parathion: 0,0-diethyl 0-p-nitrophenyl phosphorothioate	45.1%
Aromatic petroleum derivative solvent	49.4%
INERT INGREDIENTS:	5.5%
	100.0%

*Equivalent to 4.0 lbs. of 100% parathion per gallon.

Combustible: Do not store or use near heat or open flame. In case of fire, use water spray, foam, dry chemical or CO_2.

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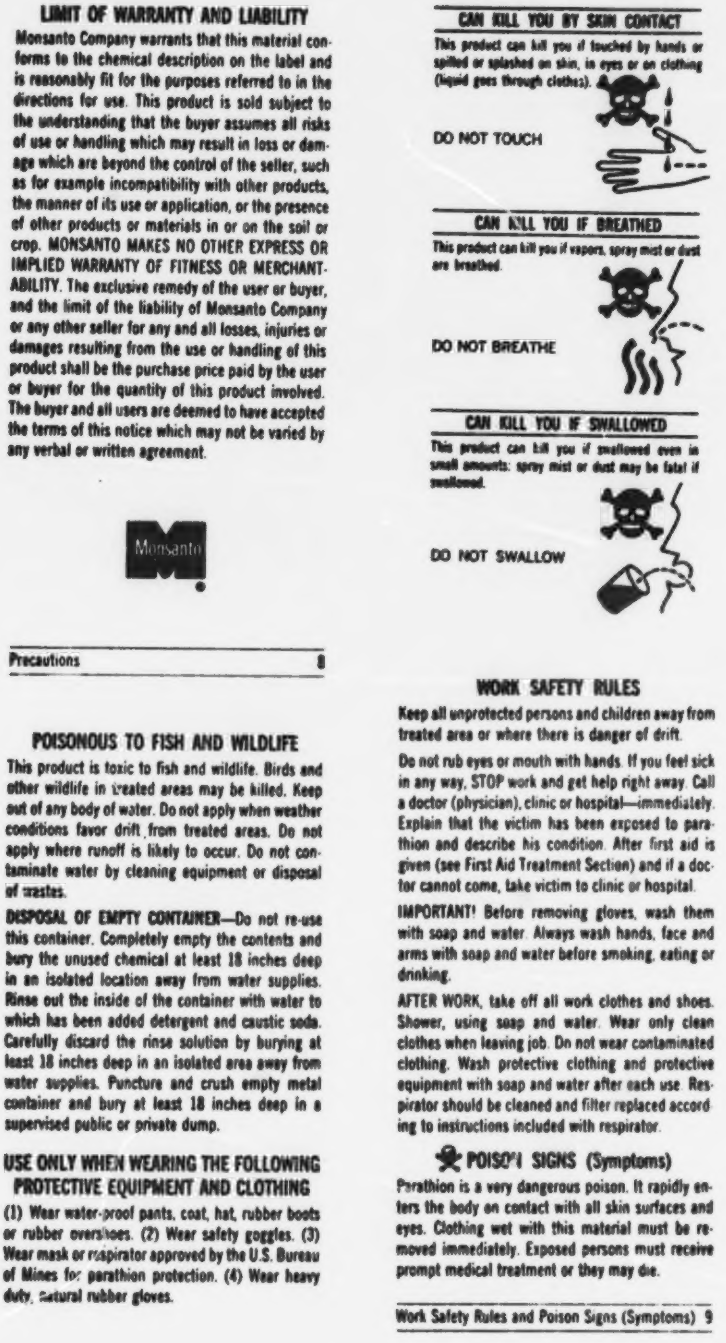
a/ Sample label of Monsanto Company, St. Louis, Missouri, EPA Reg. No. 524-132.
This formulation is not currently marketed by Monsanto, but the label is being made available to Monsanto's formulator customers for parathion technical (J. M. Magner, Communication, 10 May 1974.

Table 31. (Continued)



LIMIT OF WARRANTY AND LIABILITY

Monsanto Company warrants that this material conforms to the chemical description on the label and is reasonably fit for the purposes referred to in the directions for use. This product is sold subject to the understanding that the buyer assumes all risks of use or handling which may result in loss or damage which are beyond the control of the seller, such as for example incompatibility with other products, the manner of its use or application, or the presence of other products or materials in or on the soil or crop. MONSANTO MAKES NO OTHER EXPRESS OR IMPLIED WARRANTY OF FITNESS OR MERCHANTABILITY. The exclusive remedy of the user or buyer, and the limit of the liability of Monsanto Company or any other seller for any and all losses, injuries or damages resulting from the use or handling of this product shall be the purchase price paid by the user or buyer for the quantity of this product involved. The buyer and all users are deemed to have accepted the terms of this notice which may not be varied by any verbal or written agreement.

Monsanto

Precautions 8

CAN KILL YOU BY SKIN CONTACT

This product can kill you if touched by hands or spilled or splashed on skin, in eyes or on clothing (liquid goes through clothes).

DO NOT TOUCH

CAN KILL YOU IF BREATHED

This product can kill you if vapors, spray mist or dust are breathed.

DO NOT BREATHE

CAN KILL YOU IF SWALLOWED

This product can kill you if swallowed even in small amounts: spray mist or dust may be fatal if swallowed.

DO NOT SWALLOW

POISONOUS TO FISH AND WILDLIFE

This product is toxic to fish and wildlife. Birds and other wildlife in treated areas may be killed. Keep out of any body of water. Do not apply when weather conditions favor drift from treated areas. Do not apply where runoff is likely to occur. Do not contaminate water by cleaning equipment or disposal of wastes.

DISPOSAL OF EMPTY CONTAINER—Do not re-use this container. Completely empty the contents and bury the unused chemical at least 18 inches deep in an isolated location away from water supplies. Rinse out the inside of the container with water to which has been added detergent and caustic soda. Carefully discard the rinse solution by burying at least 18 inches deep in an isolated area away from water supplies. Puncture and crush empty metal container and bury at least 18 inches deep in a supervised public or private dump.

USE ONLY WHEN WEARING THE FOLLOWING PROTECTIVE EQUIPMENT AND CLOTHING

(1) Wear water-proof pants, coat, hat, rubber boots or rubber overshoes. (2) Wear safety goggles. (3) Wear mask or respirator approved by the U.S. Bureau of Mines for parathion protection. (4) Wear heavy duty, natural rubber gloves.

WORK SAFETY RULES

Keep all unprotected persons and children away from treated area or where there is danger of drift.

Do not rub eyes or mouth with hands. If you feel sick in any way, STOP work and get help right away. Call a doctor (physician), clinic or hospital—immediately. Explain that the victim has been exposed to parathion and describe his condition. After first aid is given (see First Aid Treatment Section) and if a doctor cannot come, take victim to clinic or hospital.

IMPORTANT! Before removing gloves, wash them with soap and water. Always wash hands, face and arms with soap and water before smoking, eating or drinking.

AFTER WORK, take off all work clothes and shoes. Shower, using soap and water. Wear only clean clothes when leaving job. Do not wear contaminated clothing. Wash protective clothing and protective equipment with soap and water after each use. Respirator should be cleaned and filter replaced according to instructions included with respirator.

☠ POISON SIGNS (Symptoms)

Parathion is a very dangerous poison. It rapidly enters the body on contact with all skin surfaces and eyes. Clothing wet with this material must be removed immediately. Exposed persons must receive prompt medical treatment or they may die.

Work Safety Rules and Poison Signs (Symptoms) 9

Table 31. (Continued)

First Aid Treatment 10

Some of the signs and symptoms of poisoning are: Headache, nausea, vomiting, cramps, weakness, blurred vision, pin point pupils, tightness in chest, labored breathing, nervousness, sweating, watering of eyes, drooling or frothing of mouth and nose, muscle spasms and coma.

✚ FIRST AID TREATMENT ✚

Call a doctor (physician), clinic or hospital immediately. Explain that the victim has been exposed to parathion and describe his condition.

If breathing has stopped, start artificial respiration immediately and maintain until doctor sees victim. **If swallowed** and victim is awake (conscious) make him vomit quickly. Induce vomiting by sticking finger down throat or by giving soapy or strong salty water to drink. Repeat until vomit is clear. Never give anything by mouth to an unconscious person. Have victim lie down and keep quiet. See doctor immediately.

In case of contact, immediately flush eyes or skin with plenty of water for at least 15 minutes while removing contaminated clothing and shoes. See doctor immediately.

ATROPINE IS AN ANTIDOTE.
CONSULT PHYSICIAN FOR EMERGENCY SUPPLY.

If symptoms or signs of poisoning include blurred vision, abdominal cramps, and tightness in the chest, do not wait for a doctor but give two atropine tablets (each 1/100 grain or 0.65 milligrams) at once. (One tablet to children under five years of age.)

NOTE TO PHYSICIAN

Antidote—administer atropine sulfate in large doses, TWO to FOUR mg. intravenously or intramuscularly as soon as cyanosis is overcome. Repeat at 5 to 10 minute intervals until signs of atropinization appear. 2-PAM chloride is also antidotal and may be administered in conjunction with atropine. DO NOT GIVE MORPHINE OR TRANQUILIZERS. Parathion is a strong cholinesterase inhibitor affecting the central and peripheral nervous systems and producing cardiac and respiratory depression. At first sign of pulmonary edema, the patient should be given supplemental oxygen and treated symptomatically. Continued absorption of the poison may occur and fatal relapses have been reported after initial improvement; VERY CLOSE SUPERVISION OF THE PATIENT IS INDICATED FOR AT LEAST 48 HOURS.

POST TREATED AREA

Consult your State Agricultural Extension Service or Experiment Station regarding posting treated areas.

Note to Physician 11

Prevent Injury 12

TO PREVENT PERSONAL INJURY AND POSSIBLE FATALITIES:

Keep all persons and animals out of treated areas for 48 hours.

Vacated areas should not be re-entered until drifting insecticide and volatile residues have dissipated.

Do not use in any manner other than recommended on this label.

To avoid excessive residues of parathion on food or forage crops, always observe the statements found under "Directions for Use," limiting the time before harvest when parathion may be applied.

If handled indoors, provide mechanical exhaust ventilation.

Do not apply or allow drift to areas occupied by unprotected humans or beneficial animals.

Do not use or store in or around the home. Keep out of reach of children and domestic animals. Do not store near food or feed products. Bury spillage; clean up area with strong lye solution.

This product is highly toxic to bees exposed to direct treatment or residues on crops. Protective information may be obtained from your Cooperative Agricultural Extension Service.

DIRECTIONS FOR USE

Be sure to read the precautionary statements before using!

This product is designed for application after dilution with water and for use by trained operators using airplane or power ground equipment. The hazards and precautions for handling the product in this container are equally applicable to it after dilution with water for spray application. Add the concentrate to the spray tank while filling with water, and mix thoroughly either by means of a tank agitator or pump by-pass. For best results, thoroughly cover all surfaces to be treated with spray. Rates of application given below should not be exceeded. Never apply later than indicated to assure residue levels at harvest are below tolerances established by the Food and Drug Administration.

Consult the State Agricultural Extension Service or Experiment Station for specific recommendations regarding application, dosage and timing of sprays. For application by ground equipment, add the desired amount of concentrate to sufficient water to apply at least 3 gallons of water per acre. For application by aircraft, add the amount of concentrate desired per acre to ⅓ to 3 gallons of water consistent with crop growth and good coverage. Greater quantities of water may be required to give sufficient coverage of orchard trees.

Directions for Use 13

Table 31. (Continued)

FRUIT

DO NOT USE TREATED CITRUS PEEL FOR FOOD PURPOSES.

CITRUS (California) — Grapefruits, Kumquats, Lemons, Limes, Oranges, Tangeloes and Tangerines.

Scale — For purple, black, brown soft, California red, citricola, cottony-cushion and yellow scales, use ⅛ to ⅜ pint in 100 gallons of water applied at petal fall to prevent fruit scattering.

Other insects—Use 1 to 2 quarts in 100 gallons of water for control of the following additional insects infesting citrus: climbing cutworms, fruit tree leaf rollers, katydids, omnivorous leaf rollers, Fuller rose beetles, pink scavenger caterpillars, orange tortrix, orangeworms and Western tussock moths. Do not use more than 2½ gallons of this product per acre up to 30 days of harvest. Do not use more than 1 gallon of this product per acre from 30 days up to 15 days of harvest. Consult agricultural experimental authorities for specific recommendations in your area.

CITRUS (Areas other than California)—Grapefruits, Kumquats, Lemons, Limes, Oranges, Tangeloes and Tangerines.

Treat for mealybugs; chaff, cottony-cushion, Glover's, purple, Florida red, yellow, snow scales; aphids; orange dog and plant bugs, using ⅛ to ⅜ pint in 100 gallons of water. For control of mites and whiteflies, use ⅛ to ⅜ pint with 1 gallon of emulsive oil concentrate in 100 gallons of water. For controlling grasshoppers, use 1 pint per acre. Thorough coverage is essential for best results. Do not use more than 2½ gallons of this product per acre up to 30 days of harvest. Do not use more than 1 gallon of this product per acre from 30 days up to 15 days of harvest.

DO NOT APPLY TO CURRANTS AND FIGS WITHIN 30 DAYS OF HARVEST.

CURRANTS—For control of currant aphids, use ½ to 1⅜ pints per acre. For control of two-spotted spider mites, use 1 to 1⅜ pints per acre. For currant borers, use 1⅜ pints per acre.

FIGS—For two-spotted and Pacific mites, use ½ to 1 pint per 100 gallons of water. For fig scales, use 1 pint per 100 gallons of water. Do not use more than 2½ quarts of this product per acre.

DO NOT APPLY TO AVOCADO WITHIN 21 DAYS OF HARVEST.

AVOCADO—To control banded cucumber beetles, grasshoppers, citrus root weevils, red-banded thrips, avocado lace bugs, pyriform scales, webbing worms, blossom anomala, little fire ants, greenhouse thrips and tortricids, use ⅜ pint in 100 gallons of water. To control whiteflies, use ⅜ pint with 1 gallon of emulsive oil concentrate in 100 gallons of water. To control Florida red scales, Florida wax scales, dictyospermum scales and avocado leafhoppers, use ½ pint in 100 gallons of water. To control pumpkin bugs and mealybugs, use ⅓ to ¾ pint in 100 gallons of water. To control latania scales, use ¾ pint in 100 gallons of water. Do not use more than 2½ quarts of this product per acre.

DO NOT APPLY TO THE FRUITS LISTED BELOW WITHIN 15 DAYS OF HARVEST.

CANEBERRIES (Raspberries, Loganberries, Boysenberries and Blackberries) — For control of two-spotted spider mites, use ¾ pint per acre. For control of obscure and woods weevils, use at 1 quart per acre as a post harvest application to the soil or ground cover over roots of plants. For crown borers, use at 1 quart per acre but apply to crown area and lower canes.

CRANBERRIES — For control of fireworms, fruitworms, tipworms and lecanium scales, use 1½ pints per acre.

GOOSEBERRIES—For control of currant aphids, use ½ to 1⅜ pints per acre. For control of two-spotted spider mites, use 1 to 1⅜ pints per acre. For currant borers, use 1⅜ pints per acre.

DO NOT APPLY TO THE FRUITS LISTED BELOW WITHIN 14 DAYS OF HARVEST.

APPLES—For control of, European sawflies, San Jose, Forbes or scurfy scales, mealybugs, European red and two-spotted mites, bagworms, Japanese beetles, shot-hole borers, orange tortrix and apple lace bugs, dilute ½ pint in 100 gallons of water and spray to cover foliage thoroughly. For codling moths, use ⅓ pint in 100 gallons of water, 3 to 4 applications, 10 to 14 days apart, starting 10 to 14 days after petal fall; for second and third broods, spray 1 to 3 times at 10 to 14 day intervals. For fruit tree leaf rollers, use ½ pint per 100 gallons of water at petal fall and for red-banded leaf rollers, apply ½ pint per 100 gallons of water at petal fall and at first, fifth and sixth cover spray. For plum curculio, apply at ½ pint per 100 gallons of water at petal fall and 1 or 2 additional times each 7 to 10 days apart. For grasshoppers, use ¼ pint in 100 gallons. For the following insects, ⅜ pint per 100 gallons of water is adequate: bud moths; clover, Pacific, Willamette or Schoenii mites; flea weevils; rosy, wooly and green apple aphids; leafhoppers; leaf miners; and red bugs. Certain insects, such as two-spotted Willamette mites, may require repeat treatments at 7 to 10 day intervals during the summer months. Parathion sprays may injure the foliage and fruit of McIntosh apples and related varieties, such as Cortland, Kendall, Macoun, Melba, etc., and Golden Delicious or

Table 31. (Continued)

Fruit Insects cont'd. 18

Jonathan. Consult the State Agricultural Extension Service or Experiment Station for advice on possibility of injury and safening the spray by using activated carbon. Do not use more than 1½ gallons of this product per acre.

APRICOTS—To control aphids, mites, bud moths, peach tree borers, Japanese beetles and leaf rollers, use ¾ pint per 100 gallons of water. Control of codling moths, lesser peach tree borers, grasshoppers, and tortrix requires ½ to ⅞ pint per 100 gallons. To control Oriental fruit moths, use ½ to ⅞ pint per 100 gallons of water at shuck split, 10 to 12 days later and if needed 6 and 3 weeks before harvest. For peach tree borers and lesser peach tree borers, apply 2 or 3 sprays to trunk from ground to scaffold limbs timed with moth emergence. Use ⅜ pint in 100 gallons of water for control of Pandemis moths. Avoid injury to bees by delaying spray till after full bloom. Do not use more than 3½ quarts of this product per acre.

BLUEBERRIES—For thrips, maggots, curculio and tip borers, use ½ pint in 100 gallons of water. For lecanium scales, use 1 pint per 100 gallons of water. Use before fruit sets or after harvest. Use from 100 to 300 gallons of diluted spray per acre, but do not apply more than 1⅛ pints of this product to one acre of blueberries at any application.

CHERRIES—For aphids and mites, mix ⅜ pint in 100 gallons of water. For sawflies, use ⅜ to ½ pint in 100 gallons of water. Use ½ pint per 100 gallons for thrips, cherry fruitworms, pear slugs, Pandemis moths, bud moths, cankerworms, rose chafers, San Jose scale crawlers, fruit flies and tortrix. For fruit tree leaf rollers, use ½ pint per 100 gallons of water at petal fall or shuck split; for plum curculio, use ⅓ pint per 100 gallons of water, 2 or 3 applications, 8 to 10 days apart, beginning at petal fall or shuck split; for Oriental fruit moths, use ½ pint in 100 gallons of water at shuck split and 10 to 12 days later. For Japanese beetles, use ¾ to 1 pint per 100 gallons. Do not use more than 2⅒ quarts of this product per acre.

GRAPES—For mites, aphids, mealybugs and berry moths, use ⅓ pint per 100 gallons of water. For leaf rollers, Japanese beetles and leaf folders, use ½ pint per 100 gallons of water. For false chinch bugs, use 1 pint in 100 gallons of water per acre by ground equipment or in 10 gallons of water by aircraft. For consperse stink bugs, use 1½ quarts per acre. For grape leafhoppers, use 1½ to 2½ quarts per acre. For black vine weevils, use 2½ quarts per acre. Do not use more than 1½ quarts of this product per acre after the fruit is the size of buckshot. Use 300 to 500 gallons of water per acre depending on age of vineyard and stage of plant growth.

Fruit Insects cont'd. 19

Fruit Insects cont'd. 20

PEACHES AND NECTARINES (Areas other than California)—For control of green peach aphids, use ⅜ pint in 100 gallons of water. For peach tree borers, leaf rollers, mites, catfacing insects, tarnished plant bugs, shot-hole borers, peach bark beetles, scales and bud moths, mix ½ pint per 100 gallons of water, and repeat if re-infestation occurs. For Oriental fruit moths, see under apricots. For plum curculio, use ½ pint per 100 gallons of water. In the South, treat at petal fall, 10 days later and repeat at 7 to 10 day intervals up to 3 weeks before harvest. In the North, treat 3 to 4 times, 7 to 10 days apart, beginning at shuck-off. For lesser peach tree and American plum borers and grasshoppers, use ¾ to 1 pint per 100 gallons. For peach tree borers and lesser peach tree borers, apply 2 or 3 sprays to trunk from ground to scaffold limbs timed with moth emergence. Do not apply more than 4 quarts of this material per acre at any application, and do not use more than 5 quarts per acre per year.

PEACHES AND NECTARINES (California)—Use as shown for other areas except do not apply within 21 days of harvest. Do not apply more than once after bloom. Do not apply more than 2½ quarts of this product per acre at any application, and do not use more than 5 quarts per acre between January 1 and harvest.

PEARS—For control of leaf miners, aphids, leaf rollers, grasshoppers, scales, mealybugs and certain mites, use the dosage described for those insects on apples. For pear psylla, use ⅜ pint per 100 gallons of water. For pear blister mites, pear slugs, green fruitworms and plant bugs, use ½ pint per 100 gallons of water. For codling moths, use ½ pint in 100 gallons of water in 2 to 4 cover sprays, beginning with the first cover. For plum curculio, apply ⅜ pint in 100 gallons of water at petal fall and 10 days later. Some injury may occur on Bosc pears, under some conditions. Do not use more than 3½ quarts of this product per acre.

PLUMS AND PRUNES—Apply ½ to ⅜ pint per 100 gallons of water for control of these insects: pear thrips, flower thrips, mites, aphids, leafhoppers, leaf rollers, peach tree borers, shot-hole borers, bud moths, tortrix, mealy plum lice and scales. Apply scale treatment when crawlers emerge. For plum curculio make 3 to 4 applications, beginning at petal fall, at rate of ⅜ pint in 100 gallons of water. For codling moths, use ½ to 1 pint per 100 gallons of water at petal fall and a summer application timed with moth emergence. For peach twig borers, use 1 pint per 100 gallons of water. Do not use more than 4 quarts of this product per acre.

Fruit Insects cont'd. 21

Table 31. (Continued)

Fruit Insects cont'd 22

STRAWBERRIES—To control flower thrips, crickets, flea beetles, red spider mites, aphids, Lygus bugs, leafhoppers, whiteflies and leaf rollers, use ½ to 1 pint in 100 to 150 gallons of water per acre. To control weevils, crown borers and strawberry leaf beetles, use ¾ pint in 100 to 150 gallons of water per acre. As a foliar treatment, do not use more than 1½ pints of this product per acre. To control garden symphylans, use 5 quarts in 40 gallons of water per acre as a preplant soil treatment.

DO NOT APPLY TO THE FRUITS LISTED BELOW WITHIN 7 DAYS OF HARVEST.

PINEAPPLES—For control of crickets and mealybugs, use ½ pint per 100 gallons of water, and apply 300 gallons of spray per acre.

DO NOT USE PARATHION ON OLIVES AFTER AUGUST 1.

OLIVES—For black, oleander and parlatoria scales, use 1 pint in 1½ gallons light-medium grade summer oil emulsion, or 1 gallon light-medium grade summer emulsive oil in 100 gallons of water, post-bloom.

NUTS

DO NOT APPLY AFTER HULLS OR HUSKS BEGIN TO OPEN. DO NOT FEED TREATED HULLS OR HUSKS TO LIVESTOCK.

ALMONDS—To control fruit tree leaf rollers, tent caterpillars and peach twig borers, use 1 pint per 100 gallons of water. As a dormant spray for parlatoria and San Jose scales, use 1 pint with 3 gallons dormant oil emulsion or 2½ gallons dormant emulsive oil in 100 gallons of water. Do not use more than 3 quarts of this product per acre.

FILBERTS—For apple mealybugs, filbert aphids, bud moths and spider mites, use ½ pint per 100 gallons of water. Do not use more than 3 quarts of this product per acre.

PECANS—For control of aphids, use ½ to ¾ pint in 100 gallons of water. To control mites, pecan nut casebearers and pecan leaf casebearers, use ¾ pint in 100 gallons of water. To control black and yellow pecan aphids, fall webworms and twig girdlers, use 1 quart per 100 gallons of water. Do not use more than 5¼ quarts of this product per acre.

WALNUTS—To control aphids, lecanium scales and walnut husk flies, use ½ pint in 100 gallons of water. Do not use more than 2½ gallons of this product per acre.

Nut Insects 23

Vegetable Insects 24

VEGETABLES

DO NOT APPLY WITHIN 21 DAYS OF HARVEST ON THE FOLLOWING CROPS UNLESS OTHERWISE SPECIFIED.

CELERY—To control aphids, mites, celery worms and tarnished plant bugs, use 1 pint per acre. To control leaf miners, whiteflies and leafhoppers, use 1½ pints per acre, but do not use within 30 days of harvest.

ENDIVE—To control green peach aphids and alfalfa loopers, use 1 pint per acre.

LETTUCE (Leaf and Bibb)—For aphids, armyworms up to third instar, cabbage loopers, imported cabbageworms, banded cucumber beetles and Lygus bugs, use ½ to 1 pint per acre. To control six-spotted leafhoppers, use ¾ pint per acre. For harlequin bugs and vegetable weevils, use 1 pint per acre. At the ½ pint rate, harvest can be made within 14 days of application.

OKRA—To control leaf miners and spider mites, use ½ to 1 pint per acre. For aphids and stink bugs, use 1 pint per acre.

SWISS CHARD—To control aphids and serpentine leaf miners, use 1 pint per acre.

DO NOT APPLY WITHIN 15 DAYS OF HARVEST ON THE FOLLOWING CROPS UNLESS OTHERWISE SPECIFIED.

BEANS—For control of bean leaf beetles and two-spotted mites, use ½ pint per acre. Use ½ to ¾ pint per acre to control thrips and lima pod borers. To control stink bugs, plant bugs, Mexican bean beetles, leaf rollers, leaf miners, leafhoppers, aphids, red spider mites and armyworms up to third instar, use 1 pint per acre.

BEETS—To control flea beetles and leaf miners, use ¾ pint per acre. For aphids, blister beetles and webworms, use 1 pint per acre. If greens are used for food, do not use within 21 days of harvest.

BLACKEYED PEAS—To control aphids, leaf miners, bean leaf rollers and stink bugs, use 1 pint per acre.

CARROTS—To control leaf miners, use ½ to ¾ pint per acre. To control leafhoppers, use ¾ pint per acre. Use 1 pint per acre to control aphids, vegetable weevils, stink bugs and petrobia mites. To control rust fly maggots (first brood), mix 1 pint with 100 gallons per acre and dribble into furrow at planting time. To control rust fly maggots (second brood), use 1 pint per acre as a foliage spray. Do not feed tops.

CUCUMBERS—For squash vine borers, aphids, cucumber beetles, leaf miners, pickleworms, mites and thrips, use ½ to 1 pint per acre. For squash bugs, stink bugs, flea beetles and leafhoppers, use 1 pint per acre. Do not apply prior to vining.

Vegetable Insects cont'd. 25

Table 31. (Continued)

Vegetable Insects cont'd. 26

EGGPLANT—To control thrips, leaf miners, blister beetles, and flea beetles, use ½ to ¾ pint per acre. To control Colorado potato beetles, use ¾ pint per acre. To control spider mites and lace bugs, use ¾ to 1 pint per acre. To control aphids, whiteflies and stink bugs, use 1 pint per acre.

GARLIC—To control onion thrips, use ½ pint per acre. To control leaf miners and petrobia mites, use 1 pint per acre.

ONIONS—To control onion thrips, use ½ pint per acre. To control onion maggot flies, use ¾ pint per acre. To control aphids, stink bugs, leaf miners, and petrobia mites, use 1 pint per acre. To control brown wheat mites, use 1¼ pints per acre.

PEPPERS—To control thrips, use 7/16 to ½ pint per acre. To control aphids, leaf miners and western potato flea beetles, use 1 pint per acre.

RADISHES—To control aphids, false chinch bugs and harlequin bugs, use ½ to 1 pint per acre. To control cabbage loopers and diamondback moths, use 1 pint per acre.

SPINACH—To control aphids, leaf miners, armyworms up to third instar, cabbage loopers, vegetable weevils, harlequin bugs, seed corn maggots, crown mites and leafhoppers, use 1 pint per acre.

SQUASH—To control cucumber beetles, aphids, stink bugs, melonworms, pickleworms, climbing cutworms, serpentine leaf miners and squash vine borers, use ½ to 1 pint per acre. To control squash bugs, flea beetles and leafhoppers, use 1 pint per acre.

SWEET POTATOES—To control aphids, spider mites, leafhoppers and stink bugs, use 1 pint per acre. To control serpentine leaf miners and morning-glory leaf miners, use 1 to 1½ pints per acre.

DO NOT APPLY WITHIN 12 DAYS OF HARVEST ON THE FOLLOWING CROPS.

SWEET CORN—To control corn earworms feeding in the bud, fall armyworms, aphids and silkflies, use ½ pint per acre. To control sap beetles and spider mites, use 1 pint per acre. To control chinch bugs, use 1½ pints per acre.

DO NOT APPLY WITHIN 10 DAYS OF HARVEST ON THE FOLLOWING CROPS UNLESS OTHERWISE SPECIFIED.

PEAS—To control aphids, pea weevils, spider mites, stink bugs, thrips, armyworms up to third instar, climbing cutworms, leaf miners, alfalfa loopers and celery loopers, use 1 pint per acre. If vines are to be used for forage, do not harvest for 15 days after treatment.

Vegetable Insects cont'd. 27

Vegetable Insects cont'd. 28

PUMPKINS—To control aphids, cucumber beetles, climbing cutworms, squash bugs and squash vine borers, use ½ to 1 pint per acre.

COLLARDS, KALE AND MUSTARD GREENS—To control aphids, leaf miners, armyworms up to third instar, cabbage loopers, vegetable weevils, harlequin bugs, seed corn maggots, crown mites and leafhoppers, use 1 pint per acre. On fall and winter crops, do not use within 15 days of harvest.

TOMATOES—To control blister beetles, use ½ pint per acre. To control hornworms, leafhoppers and psyllids, use ¾ pint per acre. For aphids, leaf miners, whiteflies, armyworms up to third instar, grasshoppers, mites, tomato russet mites, leaf-footed bugs, stink bugs, loopers and plant bugs, use 1 pint per acre.

TURNIPS—To control aphids, cabbage webworms, climbing cutworms, vegetable weevils, false chinch bugs and harlequin bugs, use ½ to 1 pint per acre. To control cabbage loopers, use 1 pint per acre. If greens are used for food, do not apply within 21 days of harvest.

DO NOT APPLY WITHIN 7 DAYS OF HARVEST ON THE FOLLOWING CROPS UNLESS OTHERWISE SPECIFIED.

ARTICHOKES—To control artichoke plume moths, use 1 quart per acre.

CABBAGE AND COLE CROPS (Broccoli, Brussels Sprouts, Cauliflower)—To control aphids, thrips, diamondback moth larvae, imported cabbageworms, cabbage loopers and armyworms up to third instar, use ½ to 1 pint per acre. To control harlequin bugs, vegetable weevils, climbing cutworms and flea beetles, use 1 pint per acre. Rates above ½ pint should not be applied to cabbage closer than 10 days until harvest.

KOHLRABI—To control aphids, use 1 pint per acre.

LETTUCE (Head)—To control aphids, cabbage loopers, imported cabbageworms, banded cucumber beetles, Lygus bugs, webworms and armyworms up to third instar, use ½ to 1 pint per acre. To control six-spotted leafhoppers, use ¾ pint per acre. For harlequin bugs, vegetable weevils and leaf miners, use 1 pint per acre. To control garden symphylans, broadcast 1¼ gallons per acre just prior to planting and thoroughly incorporate into upper 6 to 9 inches of soil.

MELONS—For squash vine borers, leaf miners and false chinch bugs, use ½ pint per acre. For aphids, leafhoppers, cucumber beetles, pickleworms and mites, use ½ to 1 pint per acre. To control thrips, squash bugs and stink bugs, use 1 pint per acre.

RUTABAGAS—To control aphids, cabbage loopers and climbing cutworms, use 1 pint per acre.

Vegetable Insects cont'd. 29

Table 31. (Continued)

Vegetable Insects cont'd 30

DO NOT APPLY WITHIN 5 DAYS OF HARVEST ON THE FOLLOWING CROP.

POTATOES—To control aphids, blister beetles, Colorado potato beetles, leaf miners, mites, plant bugs, potato psyllid, thrips, vegetable weevils and grasshoppers, use ½ to 1 pint per acre. For armyworms up to third instar, cabbage loopers, and climbing cutworms, use ¾ pint per acre. For leafhoppers, stink bugs and flea beetles, use 1 pint per acre.

FIELD AND FORAGE CROPS

DO NOT APPLY WITHIN 15 DAYS OF HARVEST, CUTTING OR FORAGE USE ON THE FOLLOWING CROPS.

ALFALFA, CLOVER, VETCH AND GRASS—For sweet clover aphids, three-cornered alfalfa hoppers, alfalfa caterpillars, and spittlebugs, use ½ pint per acre. For aphids, alfalfa weevil larvae, and adult weevils, armyworms up to third instar, clover leaf weevils, climbing cutworms, webworms, grasshoppers, crickets, spotted alfalfa aphids, leafhoppers, Lygus bugs, thrips, and tortricid moths, use ½ to 1 pint per acre. For alfalfa seed chalcids control on alfalfa grown for seed, use ½ to 1 pint per acre. California and Nevada regulations limit the use of this material to not more than ¾ pint per acre. For clover head weevils, spider mites, blister beetles, Asiatic garden beetles, sweet clover weevils, and green June beetles, use 1 pint per acre. For beet armyworms and corn earworms use 1 to 1½ pints per acre. Do not spray legumes during bloom period to avoid injury to honey bees.

PEANUTS—To control fall armyworms and red-necked peanutworms, use 1 pint per acre. To control lesser cornstalk borers, use 1 pint per acre, direct spray to soil surface and base of plants.

RICE (California)—To control rice leaf miners and tadpole shrimp, use 3/16 pint per acre. Shrimp, crabs and crayfish may be killed. Do not apply where these are importent resources.

SMALL GRAINS (Wheat, Oats, Barley)—To control armyworms up to third instar, aphids (greenbugs) and winter grain mites, use ½ pint per acre. For thrips, use ½ to ¾ pint per acre. For Say's plant bugs, use ¾ pint per acre. For black grass bugs, stink bugs, white spider mites, leafhoppers, climbing cutworms, grasshoppers and brown wheat mites, use 1 pint per acre. For chinch bugs, false chinch bugs and banks grass mites, use 1½ pints per acre.

Field and Forage Insects 31

Field and Forage Insects cont'd. 32

SOYBEANS—To control webworms, use ½ pint per acre. To control velvet bean caterpillars, green cloverworms, two-spotted mites and stink bugs, use 1 pint per acre. To control corn earworms and fall armyworms, use 1 to 1⅓ pints per acre. To control white grubs and wireworms, broadcast 1 gallon per acre just prior to planting and thoroughly incorporate into upper 4 to 6 inches of soil.

SUGAR BEETS—For alfalfa loopers, aphids, armyworms up to third instar, leafhoppers, blister beetles, flea beetles, leaf miners, Lygus bugs, stink bugs, webworms, climbing cutworms and grasshoppers, use 1 pint per acre. For false celery leaf tiers, use 1½ pints per acre. For beet crown borers, use 1½ pints per acre, ground application over the row during seedling stage. To control white grubs and wireworms, broadcast 1 gallon per acre just prior to planting and thoroughly incoporate into upper 4 to 6 inches of soil.

SUGARCANE—To control wireworms, use 2 quarts in 10 to 12 inch band in the open furrow at time of planting.

DO NOT APPLY WITHIN 12 DAYS OF HARVEST, CUTTING OR FORAGE USE ON THE FOLLOWING CROPS.

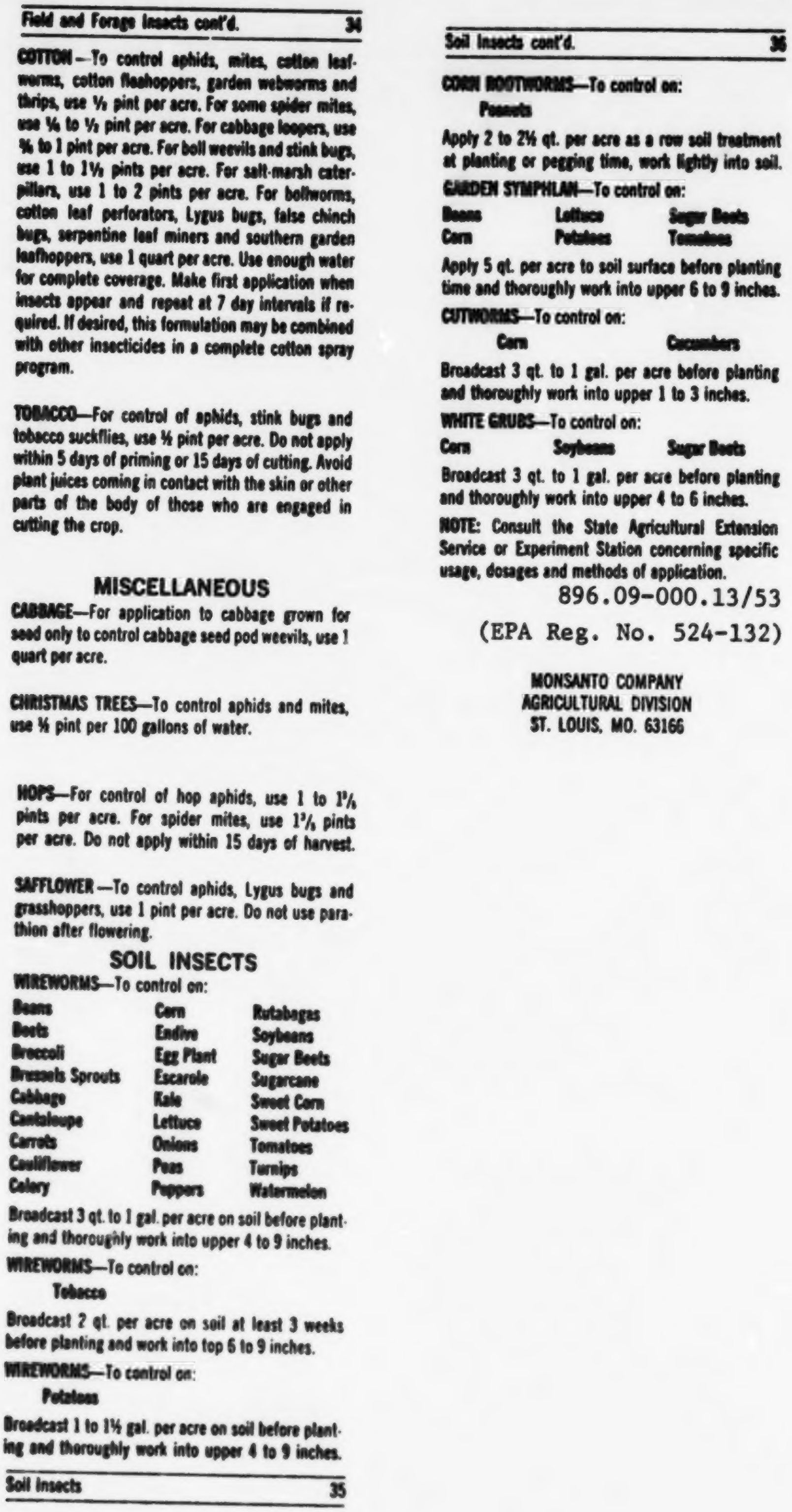
CORN—For control of European corn borers, use 1 pint per acre. Apply the first spray when 75% of the corn plants show "shot-hole" feeding and follow with two additional sprays at 5 to 7 day intervals. Use sufficient water per acre to provide complete coverage and be certain whorls of plant are well treated. For corn leaf aphids, use ½ pint per acre. For fall armyworms, corn earworms, corn rootworm adults, armyworms up to third instar, climbing cutworms, grasshoppers and Japanese beetles, use ¾ pint per acre. To control stink bugs and spider mites, use 1 pint per acre. To control chinch bugs, use 1½ pints per acre.

SORGHUM—To control sorghum midge, apply at rate of 1 pint to 1 quart per acre, 2 applications 3 to 5 days apart when approximately 90% of the heads have completely emerged from the boot or not later than start of blooming. For corn leaf aphids and mites, use ½ pint per acre. For sorghum webworms, fall armyworms, armyworms up to third instar, and corn earworms, use ¾ to 1 pint per acre. To control chinch bugs, use 1½ pints per acre. Leaf injury may occur on some hybrid varieties of sorghum. Spray a few rows a week or so before booting to test effects on plants.

DO NOT APPLY WITHIN 5 DAYS OF HARVEST ON THE FOLLOWING CROPS.

Field and Forage Insects cont'd. 33

Table 31. (Continued)



Field and Forage Insects cont'd. 34

COTTON—To control aphids, mites, cotton leafworms, cotton fleahoppers, garden webworms and thrips, use ⅓ pint per acre. For some spider mites, use ⅙ to ⅓ pint per acre. For cabbage loopers, use ⅝ to 1 pint per acre. For boll weevils and stink bugs, use 1 to 1⅓ pints per acre. For salt-marsh caterpillars, use 1 to 2 pints per acre. For bollworms, cotton leaf perforators, Lygus bugs, false chinch bugs, serpentine leaf miners and southern garden leafhoppers, use 1 quart per acre. Use enough water for complete coverage. Make first application when insects appear and repeat at 7 day intervals if required. If desired, this formulation may be combined with other insecticides in a complete cotton spray program.

TOBACCO—For control of aphids, stink bugs and tobacco suckflies, use ½ pint per acre. Do not apply within 5 days of priming or 15 days of cutting. Avoid plant juices coming in contact with the skin or other parts of the body of those who are engaged in cutting the crop.

MISCELLANEOUS

CABBAGE—For application to cabbage grown for seed only to control cabbage seed pod weevils, use 1 quart per acre.

CHRISTMAS TREES—To control aphids and mites, use ½ pint per 100 gallons of water.

HOPS—For control of hop aphids, use 1 to 1⅓ pints per acre. For spider mites, use 1⅓ pints per acre. Do not apply within 15 days of harvest.

SAFFLOWER—To control aphids, Lygus bugs and grasshoppers, use 1 pint per acre. Do not use parathion after flowering.

SOIL INSECTS

WIREWORMS—To control on:

Beans
Beets
Broccoli
Brussels Sprouts
Cabbage
Cantaloupe
Carrots
Cauliflower
Celery
Corn
Endive
Egg Plant
Escarole
Kale
Lettuce
Onions
Peas
Peppers
Rutabagas
Soybeans
Sugar Beets
Sugarcane
Sweet Corn
Sweet Potatoes
Tomatoes
Turnips
Watermelon

Broadcast 3 qt. to 1 gal. per acre on soil before planting and thoroughly work into upper 4 to 9 inches.

WIREWORMS—To control on:

Tobacco

Broadcast 2 qt. per acre on soil at least 3 weeks before planting and work into top 6 to 9 inches.

WIREWORMS—To control on:

Potatoes

Broadcast 1 to 1½ gal. per acre on soil before planting and thoroughly work into upper 4 to 9 inches.

Soil Insects 35

Soil Insects cont'd. 36

CORN ROOTWORMS—To control on:

Peanuts

Apply 2 to 2½ qt. per acre as a row soil treatment at planting or pegging time, work lightly into soil.

GARDEN SYMPHLAN—To control on:

Beans
Corn
Lettuce
Potatoes
Sugar Beets
Tomatoes

Apply 5 qt. per acre to soil surface before planting time and thoroughly work into upper 6 to 9 inches.

CUTWORMS—To control on:

Corn
Cucumbers

Broadcast 3 qt. to 1 gal. per acre before planting and thoroughly work into upper 1 to 3 inches.

WHITE GRUBS—To control on:

Corn
Soybeans
Sugar Beets

Broadcast 3 qt. to 1 gal. per acre before planting and thoroughly work into upper 4 to 6 inches.

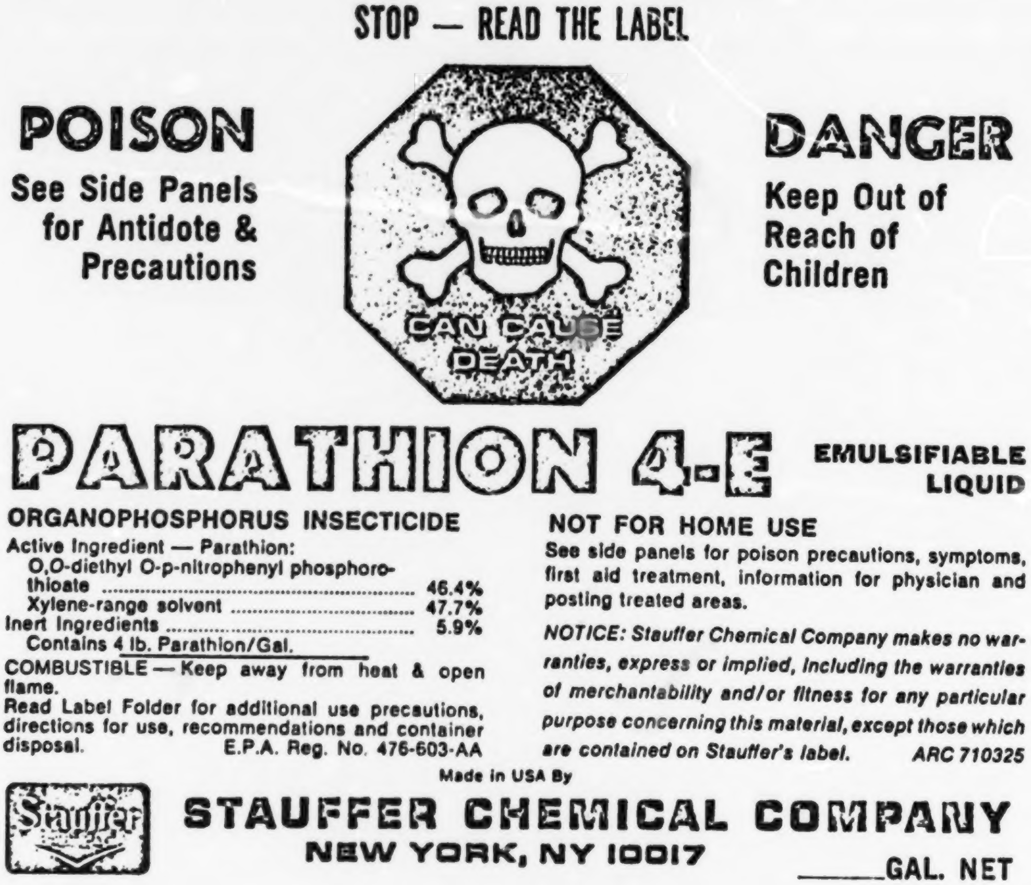
NOTE: Consult the State Agricultural Extension Service or Experiment Station concerning specific usage, dosages and methods of application.

896.09-000.13/53

(EPA Reg. No. 524-132)

MONSANTO COMPANY
AGRICULTURAL DIVISION
ST. LOUIS, MO. 63166

Table 32. REGISTERED USES OF PARATHION EMULSIFIABLE LIQUID (4 LB ACTIVE INGREDIENT PER GALLON) - CROPS AND OTHER USES, PESTS, DOSAGE RATES AND USE LIMITATIONS[a]



STOP — READ THE LABEL

POISON

See Side Panels
for Antidote &
Precautions

CAN CAUSE
DEATH

DANGER

Keep Out of
Reach of
Children

PARATHION 4-E EMULSIFIABLE LIQUID

ORGANOPHOSPHORUS INSECTICIDE

Active Ingredient — Parathion:	
O,O-diethyl O-p-nitrophenyl phosphorothioate	46.4%
Xylene-range solvent	47.7%
Inert Ingredients	5.9%

Contains 4 lb. Parathion/Gal.

COMBUSTIBLE — Keep away from heat & open flame.

Read Label Folder for additional use precautions, directions for use, recommendations and container disposal. E.P.A. Reg. No. 476-603-AA

NOT FOR HOME USE

See side panels for poison precautions, symptoms, first aid treatment, information for physician and posting treated areas.

NOTICE: Stauffer Chemical Company makes no warranties, express or implied, including the warranties of merchantability and/or fitness for any particular purpose concerning this material, except those which are contained on Stauffer's label. ARC 710325

Made in USA By

STAUFFER CHEMICAL COMPANY
NEW YORK, NY 10017

______GAL. NET

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[a] Sample label of Stauffer Chemical Company. EPA Registration No. 476-603-AA.

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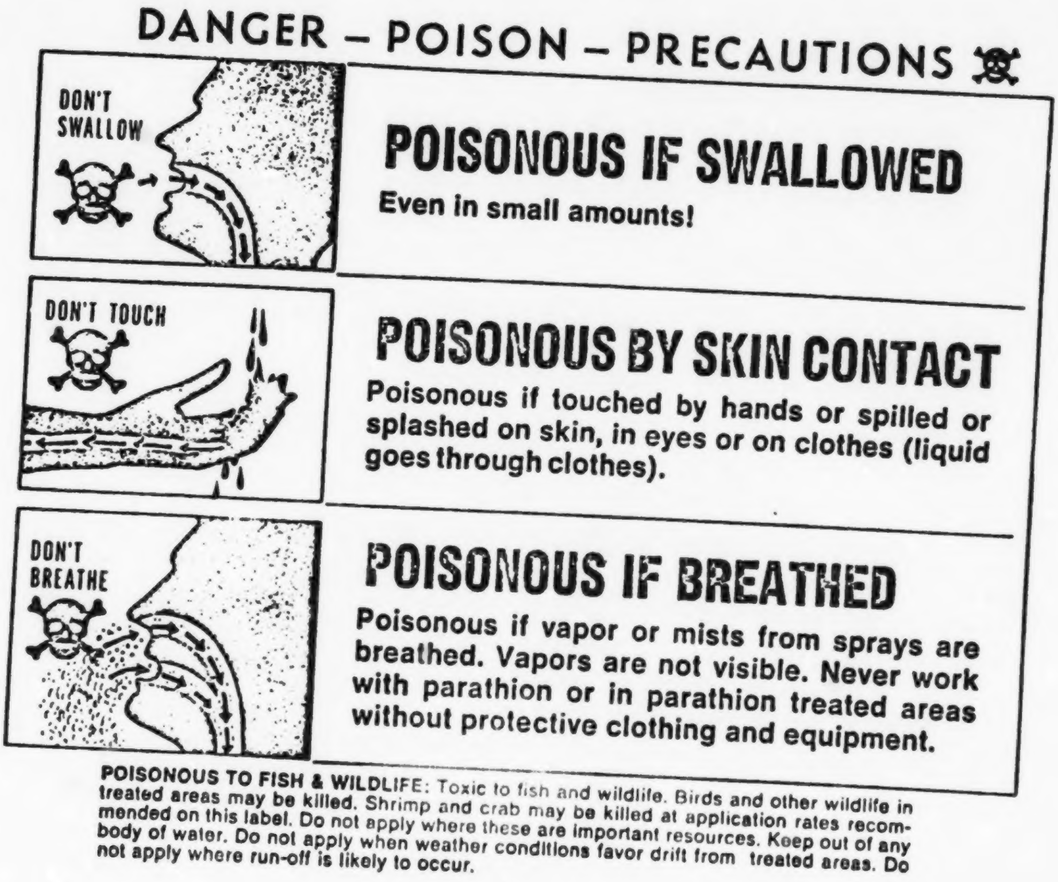
Table 32. (Continued)


DON'T SWALLOW
DON'T TOUCH
DON'T BREATHE

DANGER – POISON – PRECAUTIONS

POISONOUS IF SWALLOWED

Even in small amounts!

POISONOUS BY SKIN CONTACT

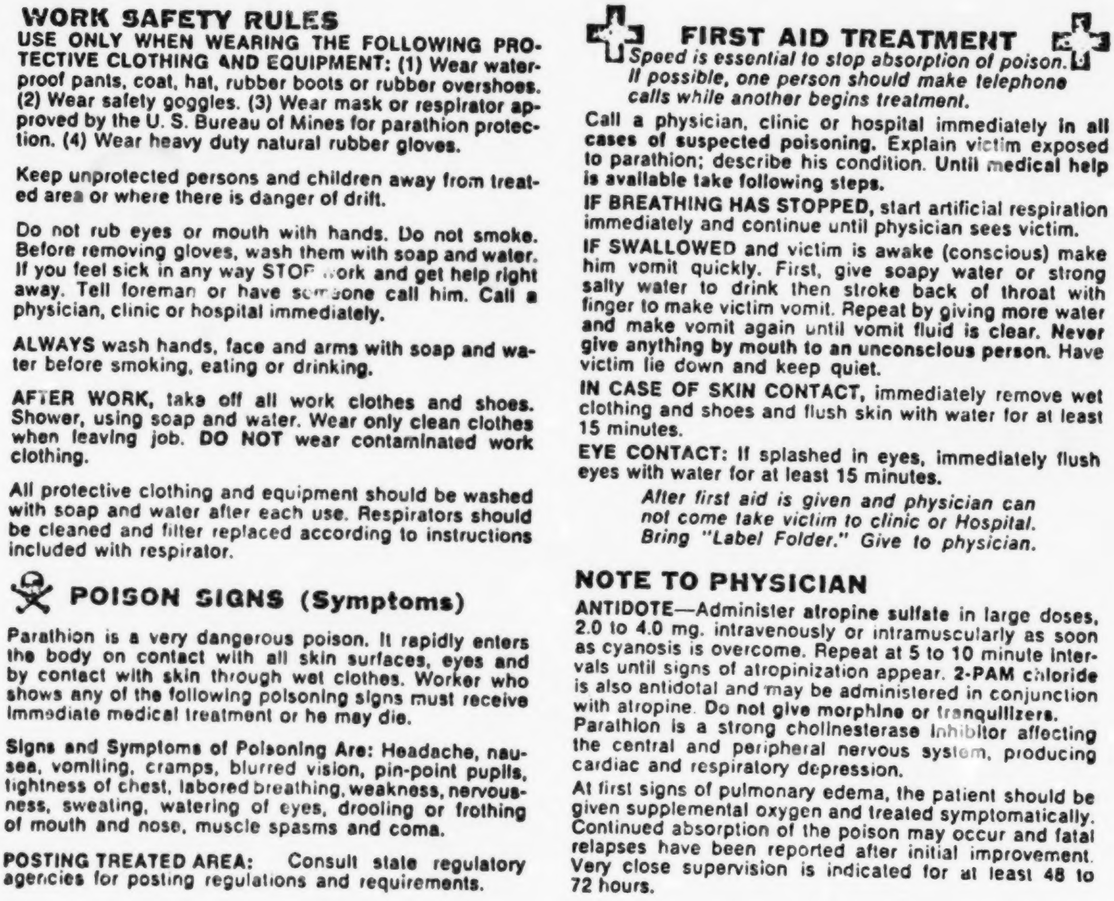
Poisonous if touched by hands or spilled or splashed on skin, in eyes or on clothes (liquid goes through clothes).

POISONOUS IF BREATHED

Poisonous if vapor or mists from sprays are breathed. Vapors are not visible. Never work with parathion or in parathion treated areas without protective clothing and equipment.

POISONOUS TO FISH & WILDLIFE: Toxic to fish and wildlife. Birds and other wildlife in treated areas may be killed. Shrimp and crab may be killed at application rates recommended on this label. Do not apply where these are important resources. Keep out of any body of water. Do not apply when weather conditions favor drift from treated areas. Do not apply where run-off is likely to occur.

Table 32. (Continued)



WORK SAFETY RULES

USE ONLY WHEN WEARING THE FOLLOWING PROTECTIVE CLOTHING AND EQUIPMENT: (1) Wear waterproof pants, coat, hat, rubber boots or rubber overshoes. (2) Wear safety goggles. (3) Wear mask or respirator approved by the U. S. Bureau of Mines for parathion protection. (4) Wear heavy duty natural rubber gloves.

Keep unprotected persons and children away from treated area or where there is danger of drift.

Do not rub eyes or mouth with hands. Do not smoke. Before removing gloves, wash them with soap and water. **If you feel sick in any way STOP work and get help right away.** Tell foreman or have someone call him. **Call a** physician, clinic or hospital **immediately.**

ALWAYS wash hands, face and arms with soap and water before smoking, eating or drinking.

AFTER WORK, take off all work clothes and shoes. Shower, using soap and water. **Wear only clean clothes** when leaving job. **DO NOT** wear contaminated work clothing.

All protective clothing and equipment should be washed with soap and water after each use. Respirators should be cleaned and filter replaced according to instructions included with respirator.

POISON SIGNS (Symptoms)

Parathion is a very dangerous poison. It rapidly enters the body on contact with all skin surfaces, eyes and by contact with skin through wet clothes. Worker who shows any of the following poisoning signs must receive immediate medical treatment or he may die.

Signs and Symptoms of Poisoning Are: Headache, nausea, vomiting, cramps, blurred vision, pin-point pupils, tightness of chest, labored breathing, weakness, nervousness, sweating, watering of eyes, drooling or frothing of mouth and nose, muscle spasms and coma.

POSTING TREATED AREA: Consult state regulatory agencies for posting regulations and requirements.

FIRST AID TREATMENT

Speed is essential to stop absorption of poison. If possible, one person should make telephone calls while another begins treatment.

Call a physician, clinic or hospital immediately **in all cases of suspected poisoning.** Explain victim exposed to parathion; describe his condition. **Until medical help is available take following steps.**

IF BREATHING HAS STOPPED, start artificial respiration immediately and continue until physician sees victim.

IF SWALLOWED and victim is awake (conscious) make him vomit quickly. First, give soapy water or strong salty water to drink then stroke back of throat with finger to make victim vomit. Repeat by giving more water and make vomit again until vomit fluid is clear. **Never give anything by mouth to an unconscious person.** Have victim lie down and keep quiet.

IN CASE OF SKIN CONTACT, immediately remove wet clothing and shoes and flush skin with water for at least 15 minutes.

EYE CONTACT: If splashed in eyes, immediately flush eyes with water for at least 15 minutes.

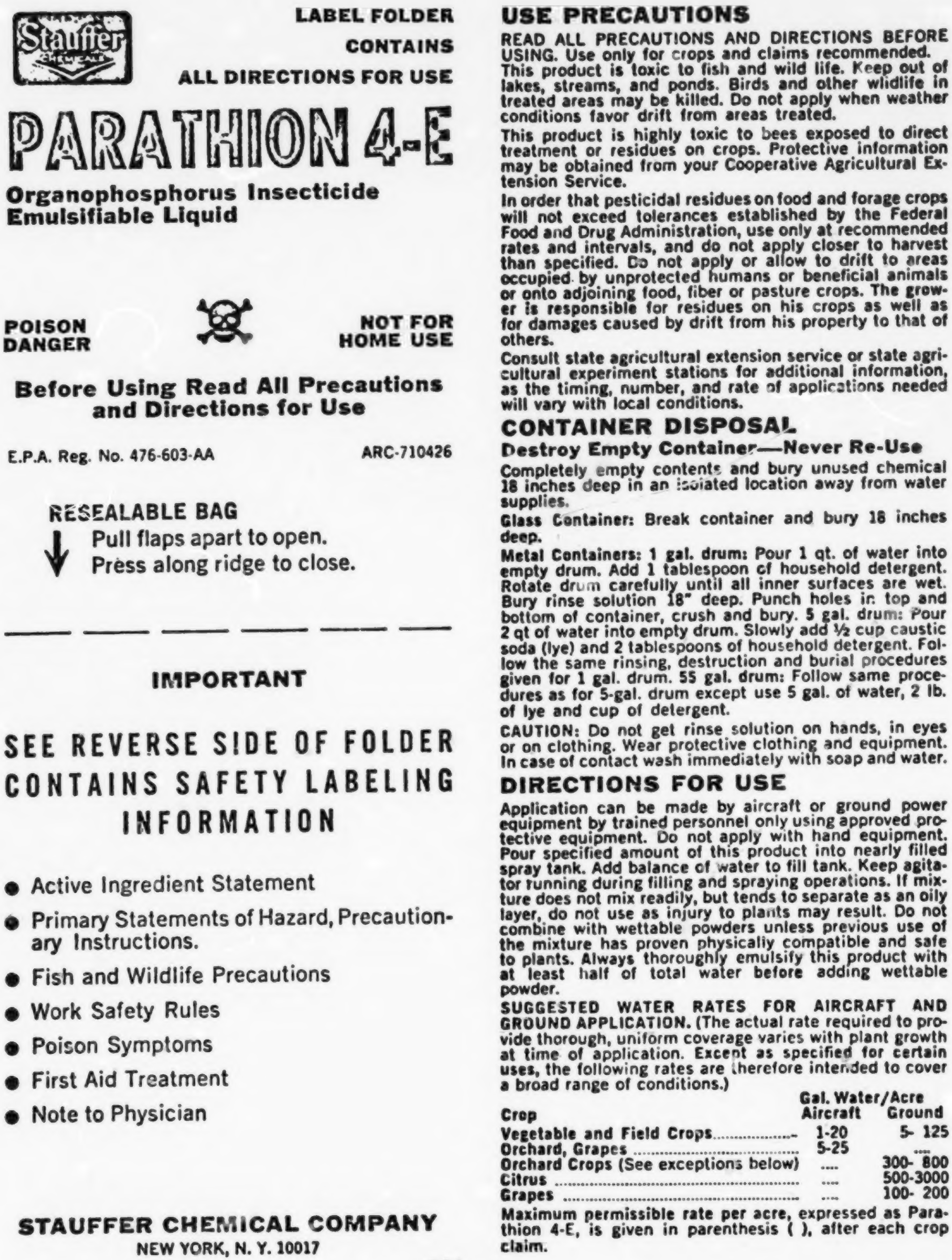
After first aid is given and physician can not come take victim to clinic or Hospital. Bring "Label Folder." Give to physician.

NOTE TO PHYSICIAN

ANTIDOTE—Administer **atropine sulfate** in large doses, 2.0 to 4.0 mg. intravenously or intramuscularly as soon as cyanosis is overcome. Repeat at 5 to 10 minute intervals until signs of atropinization appear. **2-PAM chloride** is also antidotal and may be administered in conjunction with atropine. **Do not give morphine or tranquilizers.** Parathion is a strong cholinesterase inhibitor affecting the central and peripheral nervous system, producing cardiac and respiratory depression.

At first signs of pulmonary edema, the patient should be given supplemental oxygen and treated symptomatically. Continued absorption of the poison may occur and fatal relapses have been reported after initial improvement. Very close supervision is indicated for at least 48 to 72 hours.

Table 32. (Continued)



LABEL FOLDER
CONTAINS
ALL DIRECTIONS FOR USE

PARATHION 4-E

Organophosphorus Insecticide
Emulsifiable Liquid

POISON
DANGER

NOT FOR
HOME USE

Before Using Read All Precautions and Directions for Use

E.P.A. Reg. No. 476-603-AA

ARC-710426

RESEALABLE BAG

↓ Pull flaps apart to open.
Press along ridge to close.

IMPORTANT

SEE REVERSE SIDE OF FOLDER CONTAINS SAFETY LABELING INFORMATION

- Active Ingredient Statement
- Primary Statements of Hazard, Precautionary Instructions.
- Fish and Wildlife Precautions
- Work Safety Rules
- Poison Symptoms
- First Aid Treatment
- Note to Physician

STAUFFER CHEMICAL COMPANY
NEW YORK, N. Y. 10017

USE PRECAUTIONS

READ ALL PRECAUTIONS AND DIRECTIONS BEFORE USING. Use only for crops and claims recommended. This product is toxic to fish and wild life. Keep out of lakes, streams, and ponds. Birds and other wlidlife in treated areas may be killed. Do not apply when weather conditions favor drift from areas treated.

This product is highly toxic to bees exposed to direct treatment or residues on crops. Protective information may be obtained from your Cooperative Agricultural Extension Service.

In order that pesticidal residues on food and forage crops will not exceed tolerances established by the Federal Food and Drug Administration, use only at recommended rates and intervals, and do not apply closer to harvest than specified. Do not apply or allow to drift to areas occupied by unprotected humans or beneficial animals or onto adjoining food, fiber or pasture crops. The grower is responsible for residues on his crops as well as for damages caused by drift from his property to that of others.

Consult state agricultural extension service or state agricultural experiment stations for additional information, as the timing, number, and rate of applications needed will vary with local conditions.

CONTAINER DISPOSAL

Destroy Empty Container—Never Re-Use

Completely empty contents and bury unused chemical 18 inches deep in an isolated location away from water supplies.

Glass Container: Break container and bury 18 inches deep.

Metal Containers: 1 gal. drum: Pour 1 qt. of water into empty drum. Add 1 tablespoon of household detergent. Rotate drum carefully until all inner surfaces are wet. Bury rinse solution 18" deep. Punch holes in top and bottom of container, crush and bury. **5 gal. drum:** Pour 2 qt of water into empty drum. Slowly add ½ cup caustic soda (lye) and 2 tablespoons of household detergent. Follow the same rinsing, destruction and burial procedures given for 1 gal. drum. **55 gal. drum:** Follow same procedures as for 5-gal. drum except use 5 gal. of water, 2 lb. of lye and cup of detergent.

CAUTION: Do not get rinse solution on hands, in eyes or on clothing. Wear protective clothing and equipment. In case of contact wash immediately with soap and water.

DIRECTIONS FOR USE

Application can be made by aircraft or ground power equipment by trained personnel only using approved protective equipment. Do not apply with hand equipment. Pour specified amount of this product into nearly filled spray tank. Add balance of water to fill tank. Keep agitator running during filling and spraying operations. If mixture does not mix readily, but tends to separate as an oily layer, do not use as injury to plants may result. Do not combine with wettable powders unless previous use of the mixture has proven physically compatible and safe to plants. Always thoroughly emulsify this product with at least half of total water before adding wettable powder.

SUGGESTED WATER RATES FOR AIRCRAFT AND GROUND APPLICATION. (The actual rate required to provide thorough, uniform coverage varies with plant growth at time of application. Except as specified for certain uses, the following rates are therefore intended to cover a broad range of conditions.)

Crop	Gal. Water/Acre Aircraft	Gal. Water/Acre Ground
Vegetable and Field Crops	1-20	5- 125
Orchard, Grapes	5-25	....
Orchard Crops (See exceptions below)	....	300- 800
Citrus	....	500-3000
Grapes	....	100- 200

Maximum permissible rate per acre, expressed as Parathion 4-E, is given in parenthesis ( ), after each crop claim.

Table 32. (Continued)

RECOMMENDATIONS

FRUIT AND NUT CROPS

(Also Read Directions for Use)

Unless otherwise specified, rates are given in terms of Parathion 4-E per 100 gal. of water for thorough coverage application. Unless otherwise specified, apply at the first sign of infestation and repeat at 7-10 day intervals as needed to maintain control, but observe use limitations given for specific crops.

APPLES

Codling moth, European red mite, fruit tree leaf roller, mealybug, orange tortrix, plum curculio, red-banded leaf roller (1st, 2nd and 3rd broods), two-spotted mite. Use ½ pt.

Aphids (rosy apple, green apple), bud moth, Pacific mite, red bug, Schoenii mite and Willamette mite. Use ⅜ pt.

CAUTION: Injury to fruit and foliage may result from use of this material on McIntosh and related varieties. Do not apply within 14 days of harvest. (1½ gal.)

APRICOTS

Codling moth, Oriental fruit moth, tortrix. Use ½-1⅛ pt. Bud moth, fruit tree leaf roller, spider mites, aphids. Use ¾ pt. Do not apply until danger of bee poisoning has passed. Do not apply within 14 days of harvest. (7-1/5 pt.)

CHERRIES

Black cherry aphid, eye-spotted bud moth, lesser apple worm, mites, shot-hole borer, Western cherry fruit fly. Use ½ pt. For fruit fly control, begin applications at fly emergence and repeat at 7 day intervals. Do not apply within 14 days of harvest. (4-1/5 pt.)

CITRUS

CALIF. RECOMMENDATIONS: Citrus thrips. Use 3 pt. in 10-20 gal. water when applied by aircraft; 100-200 gal. water by ground equipment. Apply at petal fall; repeat in late summer and early fall to protect new growth and prevent fruit scarring. Citrus scales. Parathion 4-E is recommended for control of California Citrus Scales. The product may be used in conventional high-pressure ground sprayers or boom sprayers and airblast equipment when the latter are capable of providing thorough coverage. **Black Scale**—Use 1 pt. Make a full coverage spray during Aug.-Nov. **California Red and Yellow scales**—Use 1¼ pt. alone or with 1½-1¾ gal. of a light or light medium oil emulsion in a full coverage spray at recommended times.

Citricola and Cottony-Cushiony scales—Use 3 pt. Apply 100-400 gal. spray per acre. Or use ⅛ pt. in a full coverage spray during Aug.-Nov.; increase dose to ¼ pt. during Feb.-Mar. and to ½ pt. during May and June applications. **Purple scale**—Use 1 pt. or ¼ pt. with 1½-1¾ gal. of a light or light medium oil emulsion in a full coverage spray at recommended times. **Max. Rate/Acre and Date Limitation:** (1 gal. up to 14 days of harvest; 2½ gal. up to 30 days of harvest).

GRAPES

Calif. Recommendations: Mealybug. Use 2 pt. plus 2 gal. dormant oil emulsion. Apply during dormant period. Spray vines after pruning but before budding. Refer to Extension bulletin for complete spray program. Leaf folder. Use 2 pt. Mites. Use ½ pt. Repeat applications at 5-7 day intervals. **Max. Rate/Acre and Date Limitation:** 3 pt. up to 14 days of harvest; 5 pt. if applied before fruit is the size of buckshot or after harvest.

NECTARINES, PEACHES

Catfacing insects, Oriental fruit moth, peach twig borer, thrips (on Nectarines only), San Jose scale. Use 1 pt. Cottony peach scale, fruit tree leaf roller, green peach aphid, spider mites. Use ⅜ pt. **Limitations: Areas other than Calif.:** Full coverage spray for control of scale insects. Do not apply within 14 days of harvest. Do not apply more than 4 qt. per acre per application or more than 5 qt. per acre per year. **Calif.:** Do not apply within 21 days of harvest. Do not apply more than once after bloom. Do not apply more than 2½ qt. per acre per application or more than 5 qt. per acre between Jan. 1st and harvest.

PEARS

Codling moth, mealybug, pear blister mite, woolly aphid. Use ½ pt. in pre-blossom spray. Pear psylla, spider mites. Use ⅜ pt. Make either pre-blossom or post-blossom sprays for the pear psylla. Under some conditions, injury may occur on Bosc pears in the Northeast. Do not apply within 14 days of harvest. (7-1/5 pt.)

PLUMS, PRUNES

Aphids, bud moth, leafhopper, leaf roller, mealy plum louse, spider mites, tortrix. Use ½ pt. Peach twig borer. Use 1 pt. Do not apply within 14 days of harvest. (1 gal.)

OLIVE

Scales (parlatoria, oleander, black). Use 1 pt. with regular oil spray during June or July. Do not apply after Aug. 1. (2¾ gal.)

STRAWBERRIES

Southeast Region: Field crickets, flea beetles, flower thrips, leaf rollers, pameras. Use ½-1 pt. Spider mites. Use ¾ pt. Do not apply within 14 days of harvest. (2 pt.) **Other Areas:** Aphids. Use 1-1-3/5 pt. Do not apply within 14 days of harvest. (1-3/5 pt.) Post-harvest control of root weevil. Use 2 pt. (2 pt.)

WALNUT

Aphids, red spider. Use ½ pt. Repeat at 7-10 days for red spider. Codling moth. Use ¾ gal. Do not apply after husks open. (2½ gal.)

VEGETABLE AND FIELD CROPS

(ALSO READ DIRECTIONS FOR USE)

Unless otherwise indicated, dosages are given in pints of Parathion 4-E per acre in sufficient water to provide thorough coverage. Begin applications when insects first appear and repeat at 7-10 day intervals as needed to maintain control, but observe use limitations given for specific crops.

ALFALFA

Alfalfa Weevil

Calif.: Apply ½-¾ pt. per acre in 15-20 gal. water per acre. Make application when 75% of terminals show feeding damage. Make no application within 15 days of cutting or forage use. (¾ pt.)

Areas Other Than Calif.: Apply ½-1 pt. per acre as specified and limited above. (1-3/5 pt.)

ALFALFA SEED CROPS

Alfalfa chalcid, alfalfa weevil larvae, aphids, armyworms, blister beetles, lygus stinkbugs. **Areas Other Than Calif.:** Use ½-1-3/5 pt.; **Calif.,** use ¾-1 pt. Use in early morning or late evening to avoid injury to pollinators. Birds and other wildlife in treated areas may be harmed. Do not apply within 15 days of harvest. Do not use Parathion if field to be cut for hay. **(Areas Other Than Calif.,** 1-3/5 pt.; **Calif.,** 1 pt.)

Table 32. (Continued)

ALFALFA, CLOVER, VETCH

Aphids (including yellow clover aphid), armyworms, blister beetles, grasshoppers. Apply ½-¾ pt. per acre. Make no application within 15 days of cutting or forage use. **(Calif.—¾ pt. for all crops)**

(Areas Other Than Calif.—1-3/5 pt. for alfalfa and vetch; 3 pt. for clover)

ARTICHOKE

Aphids, plume moth. Use 2 pt. Do not apply within 7 days of harvest. (2 pt.)

BEANS (Dry and Green)

Aphids, armyworms, leafhopper, leaf miner, leaf roller, Mexican bean beetle, spider mites, stinkbugs, whitefly. Use 1-1-3/5 pt. (1 pt.—7 days of harvest; 1-3/5 pt.—15 days.)

BEETS

Aphids, armyworms, blister beetle, flea beetle, leafhopper, leaf miner, webworm. Use 1-1-3/5 pt. Do not apply within 15 days of harvest; 21 days if treated tops are to be used for food or feed. (1-3/5 pt.)

CABBAGE, CAULIFLOWER, BROCCOLI, BRUSSELS SPROUTS, KALE, MUSTARD, TURNIPS

Aphids, armyworms, cabbage looper, flea beetle, diamond-back moth larvae, imported cabbageworm, serpentine leaf miner, thrips. Use 1 pt.

Rates and Use Limitations: Broccoli, Brussels sprouts, cauliflower: 1 pt.—7 days; 3 pt.—21 days. Cabbage, kale, mustard, turnips: ½ pt.—7 days; 1 pt.—10 days (cabbage only 3 pt.—21 days).

CARROTS

Aphids. Use ⅜-⅔ pt. per 100 gal. Apply 300 gal. per acre. Do not use treated carrot tops for food or feed. Do not apply within 15 days of harvest. (2 pt.)

CELERY

Aphids, celeryworms, celery leaf tier, leafhoppers, spider mites. Use 1-2 pt. (1 pt.—21 days of harvest; 2 pt.—30 days.)

CORN

Armyworms, corn rootworm adults (make full coverage applications to foliage when adult beetles become numerous and repeat as necessary). Use ½ pt. European corn borer. Use 1-2 pt. Do not apply within 12 days of picking or cutting for forage. (2 pt.)

COTTON

Aphids, armyworms (up to 3rd instar), brown cotton leafhopper, false chinch bug, salt-marsh caterpillar, serpentine leaf miner, southern garden leafhopper, stink bugs, spider mites, thrips. Use 1 pt.

Bollworm, cotton leaf perforator, fleahopper, lygus bugs, tobacco budworm. Use 2 pt.

Limitations. For all uses listed above do not exceed 2⅔ pt. per acre. Do not apply within 5 days of handpicking. Workers entering treated fields within 24 hours of application should wear protective clothing.

CUCUMBER, SQUASH, MELONS

Aphids, cucumber beetle, cutworms, darkling ground beetle, flea beetle, leafhopper, leaf miner, melonworm, pickleworm, serpentine leaf miner, squash bug, spider mites, petrobia mite, thrips. Use 1 pt. Do not apply unless plants are dry nor before plants start to vine. Do not apply within 15 days of harvest on cucumber and squash; 7 days of harvest on melons. (1 pt.)

EGGPLANT

Aphids, leaf miner, red spider mites, thrips. Use ⅜-½ pt. Do not apply within 15 days of harvest. (1-3/5 pt.)

LETTUCE, ENDIVE

Aphids, leafhoppers. Use 1 pt. Do not apply within 7 days of harvest on head lettuce; 21 days on endive, leaf and bibb lettuce. (1 pt.)

MOSQUITO CONTROL

In Rice Fields and Irrigated Pastures: Apply 3.2 fluid ounces in 5-20 gal. water by aircraft or in 25-100 gal. water by ground equipment. Do not apply to water drainage areas where run-off or flooding will contaminate ponds, lakes or streams. Keep out of tidal marshes and estuaries. Do not apply with 1 day of harvest of rice; 7 days of pasturing or harvest of grass pasture. (3.2 fluid ounces)

OKRA

Aphids, flea beetle, leaf miner, stinkbugs. Use ½-1 pt. Do not apply within 21 days of harvest. (1-3/5 pt.)

ONIONS

Thrips: Apply 1-1-3/5 pt. and repeat at weekly intervals as necessary for control. Do not apply within 15 days of harvest. (1-3/5 pt.)

PEAS

Aphids, armyworms, climbing cutworms, pea weevil, serpentine leaf miner, thrips: Use 1 pt. Do not apply within 10 days of harvest. (1 pt.)

PEPPERS

Aphids, pepper maggot, flea beetle, serpentine leaf miner, thrips. Use 1-1-3/5 pt. Do not apply within 15 days of harvest. (1-3/5 pt.)

POTATOES

Aphids, armyworms, Colorado potato beetle, flea beetle, green stinkbug, leafhopper, leaf miner, spider mites. Use 1-2 pt. Do not apply within 5 days of harvest. (2 pt.)

RICE

(CALIF. ONLY) Rice leaf miner, tadpole shrimp. Use 1/5 pt. in 5-10 gal. water. Apply by aircraft at the first sign of infestation after planting. Restrict spill from rice fields for 2 days following application. Do not apply within 1 day of harvest. **Caution:** Do not use within 14 days of application of Stam F.34 or Rogue. Injury may result. Do not spray over canals or laterals. (1/5 pt.)

SMALL GRAINS

(BARLEY, OATS, WHEAT)—Aphids, armyworms, grasshoppers, greenbug. Use ½-¾ pt. Do not apply within 15 days of harvest. (3 pt.)

SORGHUM

False chinch bug, grasshoppers. Use ½-⅝ pt. Corn earworm, sorghum webworm. Use ½-1 pt. Sorghum midge. Use 1 pt. and make 2 applications 3-5 days apart when approximately 90% of the heads have emerged from the boot. Aphids, mites. Use 2 pt. Do not apply within 12 days of harvest or cutting for forage. (2 pt.)

SPINACH

Aphids, leaf miner. Apply ½-1 pt. Do not apply within 14 days of harvest. (1 pt.)

SUGAR BEETS

Aphids, armyworms, blister beetle, flea beetle, leafhopper, leaf miner, mites, webworm. Use 1-1-3/5 pt. Do not apply within 15 days of harvest. (1-3/5 pt.)

TOMATOES

Aphids, flea beetle, leafhopper, serpentine leaf miner, spider mites, stinkbugs, tomato pinworm. Use 1½-2 pt. Do not apply within 10 days of harvest. (2 pt.)

One quart of parathion 4 lb/gal formulation contains 1 lb of active ingredient; 1 pt contains 0.5 lb of active ingredient. Thus, recommendations given in terms of quarts are equivalent to pounds of active ingredient; recommendations given in terms of pints convert into active ingredient by multiplication with the factor 0.5.

Parathion is also available to users in the form of emulsifiable liquids containing 2, 6 and 8 lb of active ingredient per gallon; furthermore, in the form flowable liquids containing 4 or 8 lb of active ingredient per gallon; as wettable powders containing 15 or 25% of active ingredient, and as dusts, granulars and pressurized sprays of various active ingredient concentrations. In addition, a number of dry and liquid formulations combining parathion with other insecticides and/or fungicides are registered.

For most registered uses of parathion, the rate of active ingredient recommended per acre or per volume of spray for a given use is the same, regardless of the type of formulation in which the product is applied.

State Regulations - In many of the states that currently regulate the use of pesticides, parathion is subject to use restrictions. For instance, in California, parathion is one of 42 pesticides that have been designated as "injurious or restricted materials." The use of pesticides in this category is subject to special restrictions under regulations administered by the State Department of Agriculture. A permit from the County Agricultural Commissioner must be obtained for the use of parathion. The product may not be applied in any location where damage, illness or injury appears likely to result through direct application, drift, or residue, to persons, other crops, or animals (including honeybees) other than the pest(s) which the application is intended to destroy.

Before parathion is applied, the person responsible for the application must give warning to all persons known to be on the property to be treated. After any formulation containing parathion has been applied at a rate greater than 1 lb of active ingredient per acre, the treated property must be posted for 2 weeks to provide adequate warning to persons at the point or points of normal entry. The warning notice must be readable at a distance of 25 ft.

Under this code, it is unlawful to sell or deliver parathion-containing pesticide products to any person who is required to have a permit, unless the person or his agent signs a statement that he has a valid permit to use the product.

Similar restrictions on the use of parathion are in effect in a number of other states, although California appears to have the most specific and best enforced regulations. In some states, parathion can be used only by licensed applicators.

Details of the pesticide use and application laws on a state-by-state basis have been summarized by EPA, Office of Pesticides Programs, in a publication entitled "guide for Analyzing Pesticide Legislation/Digest of State Pesticide Use and Application Laws." This pesticide law digest is being kept up to date by addition and replacement pages issued to holders from time to time.

In addition to restrictions on the use of parathion imposed by state statutes and regulations, many states augment parathion product label requirements with specific recommended uses designed to accommodate local or regional requirements. These are usually issued jointly by the State Agricultural Experiment Station and Extension Service in cooperation with the U.S. Department of Agriculture. The state insecticide use recommendations are issued or revised annually.

Production and Domestic Supply of Parathion in the United States

Volume of Production - The United States Tariff Commission, named three basic producers of parathion in the United States: Monsanto Co., Stauffer Chemical Co., and Kerr-McGee Chemical Corp.[1] Parathion is currently produced by Monsanto.

In that report, the production and sales volumes of parathion are not reported individually. Parathion is included in a group consisting of seven other specified, and additional unspecified cyclic phosphorothioates and phosphorodithioates. The reported production volume for this entire group in 1972 was 44,385,000 lb of active ingredients. Through a process of careful analysis of the use patterns of all organophosphate insecticides in this group, supported by information from trade sources, Midwest developed estimates on the volume of production of all major products in the group. The estimated volume of production of parathion in 1972 is 14 million pounds of active ingredient.

1/ U.S. Tariff Commission, Synthetic Organic Chemicals, U.S. Production and Sales, 1972, TC Publication 681 (1973).

According to Midwest Research Institute/RvR Consultants, 1974[1] about 10 million pounds of this total were produced by Monsanto Company, the balance by Stauffer and Kerr-McGee.

Imports - Imports of pesticides that are classified as "benzenoid chemicals" (including parathion) are reported by the U.S. Tariff Commission in its annual report on "Imports of Benzenoid Chemicals and Products" (TC 1973, and covers the calendar year 1972. According to this source, there were no imports of parathion into the United States in 1972.

Exports - Pesticide exports are reported by the Bureau of the Census in its annual report FT 410. Technical (unformulated) parathion and methyl parathion are included in this report in Schedule B, Section 512.0652. Formulations of parathion (and of all other cyclic and acyclic organic phosphate insecticides) are included in Schedule B, Section 599.2035, entitled "Organic phosphate containing pesticidal preparations, except household and industrial and except fly sprays and aerosols."

Total exports of organic phosphate insecticides in these two categories for 1972 are as follows:

Section 512.0652 (parathion and methyl parathion technical) - 16,533,940 lb

Section 599.2035 (organic phosphate containing formulations) - 15,898,884 lb

To derive the 1972 export volume of parathion from these totals, Midwest Research Institute made a thorough analysis of these two pesticide export categories by unit dollar values and by countries of destination. This information was then matched against the crop protection problems and the pesticide trading patterns of the countries of destination. Additional information was obtained from confidential trade sources. Estimates from this data place the 1972 export volume of parathion at 4 million pounds of active ingredient.

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[1] Midwest Research Institute/RvR Consultants, "Production, Distribution, Use, and Environmental Impact Potential of Selected Pesticides," (draft), Council on Environmental Quality, Contract No. EQC-311 (15 March 1974).

Domestic Supply - The information presented in the preceding three sections estimates the domestic supply of parathion in the United States in 1972. Subtracting exports from total production, Midwest Research Institute concludes that about 10 million pounds of active ingredient were used domestically in 1972.

It is not possible at this time to make comparable estimates for 1973, because the U.S. Tariff Commission report on the production and sales of pesticides and related products in 1973 will not become available until the late fall of 1974.

Formulations - Parathion is available to users in the United States in a variety of different formulations, and through a considerable number of suppliers. The basic domestic producers of technical parathion sell a large share, or all of their production, as technical to formulator-customers who prepare and sell different formulations of parathion under a variety of different labels and brand names to end users, either directly, or through wholesalers, and/or retailers.

Frear (1972)[1] lists almost 100 products containing parathion, offered by many different suppliers, as follows:

	Products
Aerosols (concentration not given)	2
Sprays (emulsifiable liquids containing 2, 4, 6, or 8 lb of AI per gallon; flowable liquids containing 4 or 8 lb of AI per gallon; wettable powders containing 15 or 25% of AI)	60
Dusts (varying concentrations)	22
Granulars (varying concentrations)	4
Manufacturing concentrates	4

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[1] Frear, D. E. H., Pesticide Handbook Entoma, 24th Edition, College Science Publishers, State College, Pennsylvania (1972).

In addition to these formulations containing parathion only, the product is also offered in combination formulations with other insecticides and/or fungicides, in dry and liquid formulations. Among these, an emulsifiable liquid containing 6 lb of parathion and 3 lb of methyl parathion per gallon has recently gained in popularity and use volume.

Use Patterns of Parathion in the United States

General - Essentially all of the quantities of parathion used in the United States were in agriculture. There were no significant uses of parathion by industrial, commercial or institutional pesticide users; by Federal, state, county, local, or other governmental agencies; or by home and garden users.

Surveys on the use of pesticides by farmers were conducted by the U.S. Department of Agriculture in 1964 (Agricultural Economic Report No. 131, published in January 1968), in 1966 (Agricultural Economic Report No. 179, published in April 1970), and 1971 (Agricultural Economic Report No. 252, in press). Data on the uses of parathion in 1972 were obtained by RvR Consultants.

The following farm uses of parathion were reported:

Year	Source	Farm Use
1964	USDA	6,426,000 lb of active ingredient
1966	USDA	8,425,000 lb of active ingredient
1971	USDA	9,481,000 lb of active ingredient
1972	RvR	10,000,000 lb of active ingredient

These figures indicate an upward trend in the quantities of parathion used by farmers during the 8-year period covered by the surveys.

As outlined earlier in this subsection, parathion has a very broad spectrum of effectiveness and is registered and recommended in the United States for use on a large number of fruit, nut, vegetable and field crops.

Table 33 presents a breakdown of the estimated uses of parathion in the United States in 1972, by regions and major crops. The following information sources were used in arriving at these estimates:

1. The three USDA surveys of pesticide uses by farmers mentioned above.
2. The annual USDA publication "Pesticide Review" (Agricultural Stabilization and Conservation Service).
3. Results of a survey of the Federal/State Cooperative Extension Services in all 50 states and in Puerto Rico conducted by RvR Consultants in 1973.
4. Analyses of state pesticide use recommendations.
5. Local and regional estimates on pesticide use volumes obtained from State Research and Extension personnel in personal communications.
6. Pesticide use reports from the States of Arizona, California, Illinois, Indiana, Michigan, Minnesota, and Wisconsin.
7. Data on pesticide uses supplied by the EPA Community Pesticide Studies Projects in Arizona, Hawaii, Idaho, Mississippi, South Carolina, Texas, and Utah.
8. Estimates and information obtained from basic producers of parathion and other pesticides, and from pesticide trade sources.
9. Pesticide use surveys conducted recently by Wallaces' Farmer, Des Moines, Iowa; Prairie Farmer, Chicago, Illinois; and Wisconsin Agriculturist, Madison, Wisconsin.
10. "Agricultural Statistics," an annual publication of the U.S. Department of Agriculture.

Data from all of these numerous and diverse sources were carefully analyzed, correlated cross-checked and cross-validated. The resulting estimates as summarized in Table 33 are believed to be the best and most up-to-date information on the use patterns of parathion in the United States currently available.

Table 33. ESTIMATED USES OF PARATHION ON THE U.S. BY REGIONS AND MAJOR CROPS (1972)

	Crop								
Region	Cotton	Corn	Other grains, sorghum	Vegetables	Citrus fruits	Other fruits, nuts	Tobacco	All other crops	Totals, all uses
			Thousands of Pounds of Active Ingredient						
Northeast[a]	--	Negl.	Negl.	100	--	250	Negl.	50	400
Southeast[b]	500	150	Negl.	100	100	100	300	150	1,400
East North Central[c]	Negl.	200	Negl.	100	--	100	Negl.	100	500
West North Central[d]	Negl.	200	1,050	Negl.	--	Negl.	--	50	1,300
East South Central[e]	700	50	Negl.	50	--	100	--	100	1,000
West South Central[f]	600	Negl.	1,100	400	Negl.	300	--	100	2,500
Northwest[g]	--	Negl.	50	200	--	150	--	100	500
Southwest[h]	500	Negl.	50	650	200	650	--	350	2,400
Totals, 50 States	2,300	600	2,250	1,600	300	1,650	300	1,000	10,000

Source: RvR estimates, see text.

[a] New England States, New York, New Jersey, Pennsylvania

[b] Maryland, Delaware, Virginia, West Virginia, North Carolina, South Carolina, Georgia, Florida

[c] Ohio, Indiana, Illinois, Michigan, Wisconsin

[d] Maine, Iowa, Missouri, North Dakota, South Dakota, Nebraska, Kansas

[e] Kentucky, Tennessee, Arkansas, Louisiana, Mississippi, Alabama

[f] Oklahoma, Texas

[g] Montana, Idaho, Wyoming, Colorado, Utah, Washington, Oregon, Alaska

[h] New Mexico, Nevada, Arizona, California, Hawaii

Parathion Use Patterns by Regions - Analyzing parathion uses on a geographical basis, it is interesting to note that about one-half of the total quantity of the product used in the U.S. in 1972 (almost 5 million pounds AI) was used in the Southwestern United States, i.e., in the West South Central and Southwest regions (Table 33). In these areas, about 1.2 million pounds of parathion AI were used on grain crops other than corn (primarily sorghum); 1.1 million pounds AI on cotton; and about 1 million pounds AI each on vegetable and fruit crops.

The Southeastern states used an estimated 1.4 million pounds of parathion AI. Of this total, about 500,000 lb was for cotton, another 300,000 lb for tobacco. The balance was used on vegetables, citrus and other fruits, and other crops.

An estimated 1.3 million pounds of parathion AI were used in the West North Central region, primarily on sorghum and other small grains.

In the East South Central region, a combined total of about 1 million pounds of parathion AI was used on all crops, primarily on cotton (700,000 lb).

The Northeastern, East North Central and Northwestern regions of the country each used an estimated 500,000 lb of parathion AI or less. There were no single major uses on individual crops in these areas.

Parathion Use Patterns by Crops - Analyzing the parathion use pattern by crops, it appears that cotton is the single largest "consuming" crop. Parathion is registered and recommended against 19 different insect and mite pests on cotton.

The use of parathion on grain crops, especially sorghum, has increased substantially during the last few years and is almost equal to the volume used used on cotton in 1972. Increased use is largely due to infestation of the greenbug, *Schizaphis graminum*. This insect, recorded in the United States as early as 1882, for the first time severely damaged sorghum crops in a number of states in 1968. This development has been attributed to a new biotype of the species, known as "Biotype C".[1] The primary use of parathion to control the greenbug was on sorghum in Southern Nebraska, and in Kansas, Oklahoma and Texas.

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[1] See Hight, S. C., R. D. Eikenbary, R. J. Miller, and K. J. Starks, "The Greenbug and *Lysiphelbus testaceipes*," *Environmental Entomology* 1(2):205-209 (1972).

Parathion uses on vegetable and fruit crops each amounted to about 1.6 million pounds of active ingredient in 1972, with the largest amounts of product used in the Southwestern states.

Approximately 600,000 lb of parathion AI were used on corn in 1972; and about 300,000 lb AI each on citrus fruits and on tobacco. The remaining 1 million pounds of AI went on a large variety of other crops, with no single one outstanding.

Parathion Uses in California - The State keeps detailed records of pesticide uses by crops and commodities. They are published quarterly and summarized annually. Table 34 summarized the uses of parathion in California by major crops for the 4-year period 1970 to 1973. The total volume of parathion used during this period varied relatively little, but there were considerable changes in the uses on different crops from year to year. For instance, the use of parathion on citrus fruits increased from 137,000 lb AI in 1970 to 234,000 lb in 1973. Parathion uses on stonefruits (peaches, nectarines, apricots) varied from a low of 83,000 lb AI in 1971 to a high of 227,000 1' in 1970.

Tables 35 and 36 present parathion uses in California by crop, number of applications, pounds of active ingredient, and number of acres treated, for 1972 and 1973, the two most recent years for which such data are available. In both years, parathion was used in California on close to 100 different crops.

Incongruities and inconsistencies were found in statistics on parathion uses on California crops.

A significant amount, 90,000 lb, of parathion AI was reportedly used on cotton in California in 1972, and 107,000 lb in 1973. However, the University of California Extension Service recommends the use of parathion on cotton only against the false chinch bug, described as an "occasional insect problem," but not against major California cotton pests such as the cotton bollworm, the pink bollworm, the cotton leafperforator, Lygus bugs, or mites.

The California Department of Agriculture reported using 186,000 lb of parathion AI on citrus crops (oranges, lemons, grapefruit) in 1971 (Table 34). However, the U.S. Department of Agriculture in Quantities of Pesticides Used by Farmers in 1971 (Agricultural Economic Report No. 252; in press) reports only 68,000 lb of parathion used on citrus fruits in the entire U.S. During the 1971 to 1972 season, California produced about 22% of all citrus fruits in the United States.[1]

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[1] U.S. Department of Agriculture, Agricultural Statistics, 1973, Table 314, p. 219.

Table 34.* PARATHION USES IN CALIFORNIA BY MAJOR CROPS AND OTHER USES (1970-1973)

Crop	Year 1973	1972	1971	1970
	Thousands of Pounds of Active Ingredient			
Citrus (oranges, lemons, grapefruit)	234	185	186	137
Peaches, nectarines, apricots	157	170	83	227
Plums, prunes	58	68	22	83
Almonds	82[a]	87	11	70
Olives	9	30	11	9
Tomatoes	90	55	85	62
Lettuce	90	80	180	84
Beans	30	29	23	28
Artichokes	49	60	49	19
Cotton	107	90	108	87
Sugar beets	71	48	71	54
Alfalfa	23	24	32	42
Rice	29	37	37	20
Vector control	26	20	29	51
All other uses	159	114	142	199
Totals, all uses	1,214[a]	1,097	1,069	1,172

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*California Department of Agriculture, Pesticide use reports for 1970, 1971, 1972 and 1973.

a/ Quantity used on almonds in 1973 and 1973 total reduced by 738,000 lb to adjust for apparent system error; see text.

Table 35. USE OF PARATHION IN CALIFORNIA IN 1972, BY CROPS, APPLICATIONS, QUANTITIES, AND ACRES TREATED

Commodity	Applications	Pounds	Acres
Alfalfa	762	22,750.66	67,005.20
Alfalfa fur seed	26	891.86	1,960.00
Almond	946	87,125.86	56,341.56
Anise	2	4.35	8.00
Apple	23	474.32	347.50
Apricot	330	20,234.65	14,505.75
Artichoke	1,197	59,719.59	125,272.20
Asparagus	3	187.50	75.00
Avocado	5	230.00	144.00
Barley	31	1,100.42	2,905.00
Beans, dry edible	579	27,374.78	39,437.20
Beans, green or forage	42	1,122.56	1,521.00
Beans for seed	1	15.24	20.00
Beet	145	4,087.71	9,442.68
Berries	1	6.49	7.00
Boysenberry	1	5.00	10.00
Broccoli	117	1,480.44	2,790.60
Brussels Sprouts	190	3,865.67	6,320.50
Cabbage	205	1,762.94	3,243.53
Cantaloupe	12	575.12	1,234.00
Carrot	92	2,452,39	3,950.45
Cattle lot	1	2.28	6.00
Cauliflower	219	2,268.67	4,144.70
Celery	1,005	8,375.55	16,176.43
Cherries/sweet	25	767.10	570.50
Chinese cabbage	4	3.50	7.00
Citrus	176	13,721.63	5,242.86
Clover for seed	1	148.47	80.00
Corn/field	45	4,949.58	4,619.00
Corn/sweet	45	9,970.83	4,568.00
Cotton	1,579	90,188.18	138,256.00
County or city parks		79.21	
Cucumber or pickle	13	714.06	442.00
Deciduous fruit crops	1	200.00	80.00
Eggplant	1	0.06	12.50
Endive	2	6.00	10.00
Escarole	1	3.39	6.00
Fallow (open ground)	18	818.76	479.00
Flowers	37	939.77	188.25
Foliage	2	302.23	285.10
Garlic	41	1,036.71	1,357.64
Grapefruit	42	2,893.88	518.50
Grape	131	14,475.03	5,936.00
Grasses/field	1	138.17	148.00
Honeydew melon	1	47.38	80.00
Leek	2	4.61	18.00
Lemon	120	7,738.15	3,352.66
Lettuce/head	5,259	79,298.56	143,022.99
Lettuce/leaf	111	822.90	1,797.28

Table 35.* (Continued)

Commodity	Applications	Pounds	Acres
Melons	20	882.34	1,658.00
Nectarine	848	34,638.86	16,818.24
Nonagricultural areas	86	1,215.85	12,870.00
Nursery stock	3	65.30	62.00
Oats	6	303.09	230.00
Okra	4	24.59	38.00
Olive	130	30,120.72	2,753.73
Onion/dry	434	10,505.22	16,689.57
Onion/green/spring/shallot	14	383.23	590.00
Orange	1,892	160,377.37	52,854.95
Orchard	1	15.36	58.00
Ornamentals	23	72.61	161.50
Ornamental bedding plants	6	13.92	20.00
Other agencies		86.99	
Parsley	1	0.03	12.00
Pasture/rangeland	48	764.00	8,118.50
Peach	2,018	115,217.86	63,054.65
Pear	75	10,358.27	4,440.60
Pea	102	379.61	638.00
Pepper/bell	28	315.15	672.63
Plum	632	25,781.16	11,580.05
Potato	127	6,413.46	9,218.23
Prune	411	42,035.64	23,587.33
Pumpkin	28	910.93	1,351.00
Radish	11	84.98	149.00
Residential control		0.49	
Rice	1,047	37,089.58	160,493.20
Safflower	2	14.15	54.00
School district		10.39	
Sorghum	36	6,414.95	1,884.50
Spinach	283	4,136.89	8,280.60
Squash, summer	36	713.84	1,240.00
Squash, winter	3	36.02	110.00
Strawberry	25	248.27	438.00
Sugar beets	1,379	48,317.65	107,933.88
Tangelo	5	968.74	67.00
Tangerine	4	174.68	291.00
Tomatillo/husk tomato	1	2.40	4.00
Tomato	1,467	54,824.06	85,433.63
Turf	42	4,657.58	5,365.14
Turnip	5	49.75	71.00
University of California		50.38	
Vector control		19,502.16	
Walnut	40	2,304.36	1,472.50
Water areas	1	166.37	200.00
Watermelon	13	653.56	1,158.00
Wheat	13	406.00	1,043.00
Total	24,943	1,097,091.02	1,271,111.01

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*California Department of Agriculture, Pesticide Use Report 1972.

Table 36.* USE OF PARATHION IN CALIFORNIA IN 1973, BY CROPS, APPLICATIONS, QUANTITIES, AND ACRES TREATED

Commodity	Applications	Pounds	Acres
Alfalfa	595	17,438.41	48,943.00
Alfalfa fur seed	141	5,676.99	10,017.00
Almond	753	820,076.97	42,126.24
Anise	2	10.50	21.00
Apple	17	356.13	252.50
Apricot	257	20,545.64	13,286.00
Artichoke	1,260	49,345.86	70,363.60
Avocado	5	290.00	142.00
Barley	41	3,541.15	9,816.00
Beans, dry edible	731	25,977.89	45,711.20
Beans, green or forage	3	83.14	141.00
Beans for seed	1	45.90	50.00
Beet	428	16,050.15	31,719.40
Berries	1	4.14	7.00
Broccoli	113	1,844.81	2,837.84
Brussels sprouts	104	1,730.76	2,949.50
Cabbage	186	2,061.55	3,203.60
Cantaloupe	1	8.88	20.00
Carrot	68	1,266.47	2,983.72
Cauliflower	134	1,705.68	2,625.50
Celery	923	8,338.25	14,915.76
Cherries/sweet	16	1,169.79	286.50
Chinese cabbage	13	36.41	109.00
Citrus	79	12,609.15	3,509.30
Cole crops	4	13.26	49.00
Conifer	1	0.08	5.00
Corn/field	42	10,929.58	4,560.00
Corn/sweet	23	894.18	2,372.00
Cotton	1,866	107,117.83	175,081.44
County or city parks		87.79	
Cucumber or pickle	10	397.02	178.00
Deciduous fruit crops	1	15.74	9.00
ciduous ornamental trees	1	10.50	7.00
llow (open ground)	15	16,900.93	673.00
Flowers	92	300.29	488.75
Foliage	1	0.64	32.00
Garlic	37	1,642.56	2,726.50
Grain	2	174.67	116.00
Grapefruit	50	3,017.74	616.30
Grape	160	14,683.10	7,817.50
Lemon	140	24,787.06	4,099.60
Lettuce/lead	4,723	89,729.12	153,259.37
Lettuce/leaf	61	272.47	609.68
Melons	17	517.60	1,141.00
Mustard	1	4.95	11.00
Nectarine	743	23,236.60	14,065.13
Nonagricultural areas	27	297.42	589.00
Nursery stock	4	167.33	59.75
Oats	1	66.63	150.00
Olive	186	8,863.74	3,872.49

Table 36.* (Continued)

Commodity	Applications	Pounds	Acres
Onion/dry	401	11,761.03	16,160.25
Onion/green/spring/shallot	11	92.46	119.00
Orange	2,206	194,061.11	65,776.52
Orchard	1	98.00	56.00
Ornamentals	3	5.12	12.00
Ornamental bedding plants	3	24.00	12.00
Other agencies		11,753.39	
Pasture/rangeland	15	2,086.59	1,707.00
Peach	1,904	112,936.23	63,493.38
Pear	64	6,795.70	5,471.50
Pea	202	774.99	817.25
Pepper/bell	37	1,113.73	1,794.25
Pimento	1	69.59	75.00
Plum	588	21,962.58	10,596.13
Potato	76	2,068.47	3,438.00
Prune	421	35,670.73	20,463.25
Pumpkin	10	276.17	456.00
Radish	8	147.16	250.00
Residential control		5.40	
Rice	1,453	29,104.95	217,010.70
Rutabaga	1	9.89	16.00
Safflower	16	14,674.06	2,965.00
Salsify fur seed	1	3.50	2.00
School district		33.22	
Soil (fumigation only)	5	757.64	209.00
Sorghum	62	2,368.37	4,147.00
Sorghum fur seed	1	0.12	98.50
Spinach	187	2,040.11	4,112.34
Squash/summer	17	573.03	525.00
Squash, winter	1	30.53	100.00
Strawberry	36	463.54	842.00
Structural control		0.90	
Sudangrass	3	35.16	96.00
Sugar beets	1,779	70,502.40	143,517.78
Sunflower seed	3	493.29	575.00
Sweet potato	67	2,784.45	2,889.00
Tangelo	2	43.75	30.00
Tangerine	1	60.00	20.00
Tomato	1,514	89,928.84	113,610.50
Turf	71	7,564.36	8,541.00
Turnip	2	21.34	37.00
University of California		142.55	
Vectur Control		26,445.43	
Walnut	72	6,400.90	2,368.50
Water Areas	1	0.01	2.00
Watermelon	5	369.33	600.00
Wheat	30	1,084.96	2,241.00
Total	25,361	1,951,982.53	1,373,878.02

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* California Department of Agriculture, Pesticide Use report 1973.

Regarding the California Department of Agriculture's reported figure of 820,077 lb, of parathion used on the State's almond crops in 1973, available figures for the three preceeding years show that parathion uses on almonds were considerably lower: 1970, 70,000 lbs; 1971, 11,000 lb; 1972, 87,000 lb; (see Table 34). Based on an analysis of past level of use and the number of acres treated in 1973, Midwest Research Institute believes there to be an error in the reported 1973 figure for parathion use in California and has accordingly revised the figure (See Table 34) 1/

At the present time, no other state in the union records or publishes pesticide use data in comparable detail. Limitations of time and resources did not permit development of estimates on the uses of parathion by crops and states beyond the detail provided in Table 34.

Summary - In 1972, parathion was used in the United States on the following major crops (listed in decreasing order of volume of use): cotton, sorghum and small grains; deciduous fruit and nut crops; vegetable crops, corn; citrus fruits; tobacco; and numerous other crops, each accounting for a relatively small share of the total use.

By geographic regions, the use pattern of parathion in 1972 was as follows (regions listed in decreasing order of volume of use): West South Central (Oklahoma and Texas); Southwest (California, Arizona, Hawaii, New Mexico, and Nevada); Southeast; West North Central states;East South Central states; Northwest; Northeast.

Slightly more than 10% of the total quantity of parathion used in the U.S. in 1972 was in California, on almost 100 different crops.

1/ In a personal communication, the California Department of Agriculture agreed that the reported figure of 820,077 lb was most likely erroneous. The agency did not account for the discrepancy.

PART III. MINIECONOMIC REVIEW

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This section contains a general assessment of the efficacy and cost effectiveness of parathion. Data on the production of parathion in the United States as well as an analysis of its use patterns at the regional level and by major crop are found in Part II of this report.

Introduction

The efficacy and cost effectiveness of a specific pesticide should be measurable in terms of the increased yield or improved quality of a treated crop which would result in a greater income or lower cost than would be achieved if the pesticide had not been used. Thus, one should be able to pick an isolated test plot of a selected crop, treat it with a pesticide, and compare its yield with that of a nearby untreated test plot. The difference in yield should be the increase due to the use of the pesticide. The increased income (i.e., the yield multiplied by the selling price of the commodity) less the additional cost (i.e., the pesticide, its application and the harvesting of the increased yield) is the economic benefit due to the use of the pesticide.

Unfortunately, this method has many limitations. The data derived is incomplete and should be looked on with caution. Midwest Research Institute's review of the literature and EPA registration files revealed that experimental tests comparing crops treated with specific pesticides to the same crop without treatment are conducted by many of the state agricultural experimental stations. Only a few of these however, have attempted to measure increased yield and most of this effort has been directed toward just a few crops such as cotton, potatoes, and sorghum. Most other crop tests measure the amount of reduction in pest levels which cannot be directly related to yield.

Even the test plot yield data are marginally reliable, since these tests are conducted under actual field conditions that may never be duplicated again and are often not representative of actual field use. Thus, yield is affected by rainfall, fertilizer use, severe weather conditions, soil type, region of the country, pesticide infestation levels and the rate, frequency and method of pesticide application.

Because of these factors, yield tests at different locations and in different years will show a wide variance ranging from a yield decline to significant increases. For example, in a year of heavy pest infestation, frequent pesticidal use can result in a high yield increase because the crop from the untreated test plot is practically destroyed. Conversely, in a year of light infestation, the yield increase will be slight.

Thus, the use of test plot yield data is at best qualitative and is used for order-of-magnitude economic cost and benefit determination.

The use of market price to estimate the value received by the producer also has its limitations. If the use of the pesticide increases the yield of a crop and the national production is increased, then the market price should decline. According to J. C. Headley and J. N. Lewis (1967),[1] a 1% increase in quantity marketed has at times resulted in a greater than 1% decrease in price. Thus, the marginal revenue from the increased yield would be a better measure of value received.

A third limitation to the quantification of the economic costs and benefits is the limited availability of data on the quantities of the pesticide used by crop or pest, the acres treated, and the number of applications. In most cases the amount of parathion used on each crop or each pest is not available.

As a result of these limitations an overall economic benefit by crop or pest cannot be determined. This review presents a range of the potential economic benefits derived from the use of parathion for control of a specific pest on a specific crop. This economic benefit or loss is measured in dollars per acre for the highest and lowest yield developed from experimental tests conducted by the pesticide producers and the state agricultural experimental stations. The high and low yield increases are multiplied by the price of the crop and reduced by the cost of the parathion applied to generate the range of economic benefits in dollars per acre.

Efficacy and yield changes due to the use of parathion have been reported on several crops and pests. This report summarizes the results of these tests which include the bollworm, boll weevil, and tobacco budworm on cotton; the sorghum midge and greenbug on sorghum; the greenbug on wheat; the southern corn rootworm on peanuts; the Pacific wireworm, potato aphid and potato leafhoppers on potatoes; the European corn borer and western corn rootworm on corn; the lygus lesperus on lima beans; the tarnished plant bug on strawberries; and the pea aphid on peas.

1/ Headley, J. C., and J. N. Lewis, The Pesticide Problem: An Economic Approach to Public Policy, Resources for the Future, Inc., pp. 39-40, (1967).

Cotton

Parathion is registered for a wide variety of cotton insects. The tobacco budworm, bollworm, boll weevil, aphids, fleahoppers, leaf hoppers, cabbage loopers, spider mites, and thrips are major cotton pests treated with parathion. Application rates vary from 0.25 to 1.0 lb/acre, depending upon the type of insect. The number of applications depends upon the degree of infestation. Repeated applications are recommended for the bollworm, budworm, and boll weevil until adequate control is achieved.

Efficacy Against Pest Infestation - Data is available on the efficacy of parathion for control of the budworm, bollworm, and boll weevil-- the three major cotton pests--'from tests conducted in Texas.

Adkisson et al. (1966)1/ compared a wide variety of insecticides for control of bollworm larvae near College Station, Texas in 1965. The use of parathion resulted in a 70% kill after 48 hr when applied at 0.5 lb/acre.

Adkisson et al. (1967)2/ conducted similar tests in 1966 and reported an 85% kill of bollworm larvae 48 hr after parathion was applied at 0.5 lb/acre. Parathion was also less effective against the budworm with an 83% kill at 0.75 lb/acre after 48 hr compared to a 97% kill for 0.75 lb/acre of methyl parathion. Against adult boll weevils, 0.25 lb/acre of parathion resulted in a 97% kill after 48 hr compared to 100% for methyl parathion at the same rate.

Wolfenbarger (1973)3/ found that tobacco budworms from a susceptible strain were 2.45 times more resistant to parathion than to methyl parathion during tests conducted in Brownsville, Texas in 1970.

1/ Adkisson, Perry L., and S. J. Nemec, "Comparative Effectiveness of Certain Insecticides for Killing Bollworms and Tobacco Budworms," Publication B-1048, Texas Agr. Exp. Sta. (1966).

2/ Adkisson, Perry L., and S. J. Nemec, "Insecticides for Controlling the Bollworm, Tobacco Budworm, and Boll Weevil," MP-837, Texas Agr. Exp. Sta. (1967).

3/ Wolfenbarger, D. A., "Tobacco Budworm: Cross Resistance to Insecticides in Resistant Strains and in a Susceptible Strain," J. Econ. Entomol., 66:292-294 (1973).

Cost Effectiveness of Pest Control - Information was found on only one test relating yield changes to parathion usage. Bost (1974)[1] summarized tests conducted between 1956 and 1973 at Stoneville, Mississippi. The results of one test in 1956 showed a 253 lb/acre gain over an untreated check when nine applications of parathion at 0.5 lb/acre were made.

The 1972 price received by farmers for cotton was 14.0¢/lb for lint. Additional income from cottonseed of 4.2¢/lb and government price supports of 12.5¢/lb brought the total income to 40.7¢/lb (*Agricultural Statistics, 1973*).[2] Parathion costs averaged $1/lb in 1972, while application costs averaged $.50 per treatment (Chambers et al., 1974).[3]

Using the above cost and price data, the additional income would amount to $102.98/acre. Subtracting the cost of parathion at $9.00/acre would result in an economic benefit of $93.98/acre when parathion was used to control boll weevils, bollworms, and tobacco budworms.

Sorghum

Parathion is registered for control of aphids, greenbugs, spider mites, sorghum webworms, and sorghum midges on sorghum. Of these, sorghum midge and the greenbug are the two insects having the greatest effect on yield.

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[1] Bost, W. M., Director, "Cooperative Extension Service Mississippi State University, Mississippi State Mississippi, Summary of Test Results at Stoneville and Verona, Mississippi, and Costs of Pesticides," personal letter to Mr. David F. Hahlen (1974).

[2] U.S. Department of Agriculture, *Agricultural Statistics 1973*.

[3] Chambers, William, and Daniel Miller, Farmland Industries, Kansas City, Missouri, conversation (1974).

Efficacy Against Sorghum Midge Infestation - In the early 1960's the sorghum midge temporarily caused serious crop losses. However, early uniform planting has restricted damage from this insect to isolated late planted fields (Cate et al., 1973).[1] Huddleston et al. (1972)[2] reported on tests of insecticides for control of the midge and concluded that parathion gave superior control of midge population with two applications of 0.5 lb/acre.

Yields from tests conducted at Plainview, Texas increased from 22 to 330 lb/acre over an untreated check as the rate and number of applications increased. However, increasing the rate to 1.0 lb/acre for two applications reduced the increase to 198 lb/acre. In another test conducted in Texas in 1961, one application of 0.5 lb parathion per acre reduced yields from the untreated check by 230 bushels per acre. The authors attributed this to the application being made at late bloom.

Cate et al. (1971)[3] evaluated several insecticides for control of the midge in Lubbock County, Texas in 1969. Parathion was applied at 0.5 lb/acre and controls measured for two and three applications. The first application was made when 50% of the grain heads were out of the boot. The results showed a 63% control with two applications and 55% control with three applications. Yields compared to an untreated check increased 236 lb/acre with two applications but declined 313 lb/acre with three applications.

Cost Effectiveness of Sorghum Midge Control - The tests of Cate et al. (1973) and Huddleston et al. (1972) contain the only data found that compared yields of parathion treated sorghum plots against an untreated check. The results of these experiments showed yield changes ranging from a loss of 313 lb/acre to an increase of 330 lb/acre.

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[1] Cate, J. R., Jr., D. G. Bottrell, and G. L. Teetes, "Management of the Greenbug on Grain Sorghum. I. Testing Foliar Treatments of Insecticides Against Greenbugs and Corn Leaf Aphids," J. Econ. Entomol., 66:945-951 (1973)

[2] Huddleston, E. W., D. Ashdown, B. Maunder, C. R. Ward, G. Wilde, and C. E. Forehand, "Biology and Control of the Sorghum Midge. I. Chemical and Cultural Control Studies in West Texas," J. Econ. Entomol., 65:851-855 (1972).

[3] Cate, J. R., Jr., and D. G. Bottrell, "Field Evaluation of Insecticide Treatments for Control of the Sorghum Midge," PR-2866, pp. 13-15, Research on Grain Sorghum Insects and Spider Mites in Texas, Texas Agr. Exp. Sta. (1971).

The price of sorghum averaged $2.25/cwt in 1972. *Agricultural Statistics 1973* and the cost of parathion was $1/lb (Chambers and Miller) cost, the economic benefits would range from a loss of $10.04/acre to a gain of $4.93/acre for control of the sorghum midge.

These tests are summarized in Table 37.

Table 37. Results of Parathion Application Sorghum Midge

Date	Application Rate (lb AI/acre)	No.	Yield Increase* (lb/acre)	Additional income* $2.25/cwt ($/acre)	Application cost at AI $1/lb + treatment at $.50/application	Economic benefit* ($/acre)	Source
1961	0.5	1	(224)	(5.04)	1.00	(5.54)	a/
1964	0.5	1	22	0.50	1.00	0	
	1.0	1	154	3.47	1.50	2.47	
	0.5	2	330	7.43	2.00	6.43	
	1.0	2	198	4.45	3.00	2.45	
1969	0.5	2	236	5.31	2.00	4.31	b/
	0.5	3	(313)	(7.04)	3.00	(8.54)	

* Data in parentheses indicates decreases in yield, income and economic benefit.

a/ Huddleston et al., op. cit. (1972).

b/ Cate et al., op. cit. (1971).

Efficacy Against Greenbug Infestation - Although there are numerous insects affecting sorghum, perhaps the greenbug causes the greatest damage. Prior to 1968 the greenbug had been found mostly in small grains such as wheat, barley, and oats. However, in 1968 a new biotype emerged and began infesting sorghum. Ward et al. (1970)[1] noted that, in 1968, 7.3 million acres became infested resulting in a production loss estimated at $20 million. Cate et al. (1973) reported that the Grain Sorghum Producers Board estimated that $14 million was spent for control of grain sorghum pests in 1970 compared with only $100,000 spent prior to 1968.

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[1] Ward, C. R., E. W. Huddleston, D. Ashdown, J. C. Owens, and K. L. Polk, "Greenbug Control on Grain Sorghum and the Effects of Tested Insecticides on Other Insects," J. Econ. Entomol., 63:1929-1934 (1970).

Parathion gives satisfactory control of greenbugs on sorghum. Daniels (1971)[1] conducted two tests at Bushland, Texas in 1969 and achieved a 79% reduction of greenbugs for 29 days in one test and an 89% reduction for 25 days in the other test. The first test consisted of 0.5 lb/acre of parathion and the second was at a 0.25 lb/acre rate. Yield increases were 112 and 42 lb/acre respectively.

Cate et al. (1973) conducted several experiments for insecticidal control of the sorghum greenbug in Lubbock County Texas in 1968, 1969 and 1970. Results showed greenbug control ranging from 65% to 97% 7 days after application of 0.5 lb/acre parathion. Seasonal control of greater than 90% was reported in some of the tests. Yield changes from the untreated check varied from increases of 32 to 1,083 lb/acre. Ward et al. (1970) also showed significant reduction of greenbugs in tests at Lubbock and New Deal, Texas in 1968. Yield increases varied widely from a low of 9 lb/acre to a high of 497 lb/acre. Similar results were found by DePew (1971)[2] and Harvey et al. (1970)[3].

Parathion appears to be phytotoxic to certain types of sorghum. Although DePew (1971) reported no evidence of phytotoxicity, Meisch et al. (1970)[4] evaluated various insecticides for phytotoxicity to six varieties of sorghum and found severe leaf damage and significant yield loss in one variety, but medium to low damage on the other varieties. Yields on the other varieties varied from a loss of 385 lb/acre compared to an untreated check to a gain of 408 lb/acre.

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[1] Daniels, N. E., "Insecticidal Greenbug Control in Grain Sorghum," PR-2868, pp. 16-20, Research on Grain Sorghum Insects and Spider Mites in Texas, Texas Agr. Exp. Sta. (1971).

[2] DePew, L. J., "Evaluation of Foliar and Soil Treatments for Greenbug Control on Sorghum," J. Econ. Entomol., 64:169-172 (1971).

[3] Harvey, T. L., and H. L. Hackerott, "Chemical Control of a Greenbug on Sorghum and Infestation Effects on Yields," J. Econ. Entomol., 63:1536-1539 (1970).

[4] Meisch, N. V., George L. Teetes, N. M. Randolph, and A. J. Bockholt, "Phytotoxic Effects of Insecticides on Six Varieties of Grain Sorghum," J. Econ. Entomol., 63:1516-1517 (1970).

Cost Effectiveness of Greenbug Control - The results of several tests in Texas and Kansas show that yield changes varied from a loss of 458 lb of sorghum per acre to a gain of 1,083 lb/acre when parathion was used to control the greenbug.

The price of sorghum averaged $2.25/cwt in 1972 (Agricultural Statistics 1973); the cost of parathion was $1.00/lb (Chambers and Miller 1974), the cost of application is $.50. At these prices and costs the economic benefits would range from a loss of $11.81/acre to a gain of $23.77/acre from the use of parathion to control the greenbug.

These tests are summarized in Table 38.

Table 38. Results of Parathion Application on Sorghum Greenbug

Date	Application Rate (lb AI/acre)	No.	Yield increase* (lb/acre)	Additional income* $2.25/cwt ($/acre)	Application cost at AI $1/lb + treatment at $.50/application	Economic benefit* ($/acre)	Sourc
1968	0.5	1	497	11.18	1.00	10.18	a/
	0.5	1	93	2.09	1.00	1.09	
1969	0.5	1	9	0.20	1.00	(.80)	
1969	1.0	2	(117)	(2.63)	3.00	(3.63)	b/
	1.0	2	(385)	(8.66)	3.00	(9.66)	
	1.0	2	(458)	(10.31)	3.00	(11.31)	
	1.0	2	(345)	(7.76)	3.00	(8.76)	
	1.0	2	385	8.66	3.00	5.66	
	1.0	2	408	8.82	3.00	5.82	
1968	0.5	1	32	0.72	1.00	0.72	c/
	0.5	1	770	17.33	1.00	16.33	
1969	0.5	1	47	1.06	1.00	0.06	
1970	0.5	1	1,083	24.37	1.00	23.37	
	0.1	1	1,083	24.37	0.60	23.77	
	0.25	1	783	17.62	0.75	16.87	
	0.5	1	1,023	23.02	1.00	22.02	
1969	0.5	1	84	1.89	1.00	0.89	
1968	0.25	1	439	9.88	0.75	9.13	d/
1969	0.25	1	42	0.95	0.75	0.20	e/
	0.5	1	112	2.52	1.00	1.52	

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* Data in parentheses indicate decreases in yield, income, and economic benefit.

a/ Ward et al., op. cit. (1970).

b/ Meisch et al., op. cit. (1970).

c/ Cate et al., op. cit. (1973).

d/ Harvey et al., op. cit. (1970).

e/ Depew, op. cit. (1971).

f/ Daniels, op. cit. (1971).

Wheat

The greenbug has been regarded as the most destructive of the aphids infecting small grains since its introduction into North America in the 19th Century.

Efficacy Against Pest Infestation - As a result of a heavy infestation of greenbugs in Texas in 1961, Daniels (1962)[1] evaluated several insecticides for their control on wheat. These tests at Bushland, Texas in 1961 resulted in satisfactory control with parathion. In four experiments averages of 88 to 92% reduction of greenbugs were achieved. Yields increased between 4.0 and 10.8 bushels/acre respectively.

Ward et al. (1972)[2] screened various insecticides at Clovis, New Mexico in 1969 and reported a 98% reduction in greenbugs after 14 days with parathion. Yields increased by 575 lb/acre or 9.57 bushels/acre.

Cost Effectiveness of Pest Control - Yield increases based upon the three tests reported above varied from 4.0 to 10.8 bushels/acre. At a price of $1.67/bushel for wheat in 1972 (Agricultural Statistics 1973) and an application cost of $.50 per treatment, a cost of $1.00/lb for parathion (Chambers and Miller, 1974), the economic benefit would range from $5.68 to $17.04/acre from control of the greenbug on wheat.

These are summarized in Table 39.

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[1] Daniels, N. E., "Insecticidal Control of the Greenbug," Progress Report 2247, Texas Agr. Exp. Sta. (1962).

[2] Ward, C. R., J. Owens, D. Ashdown, E. Huddleston, and W. Turner, "Greenbug Control on Wheat in 1967-1969," J. Econ. Entomol., 65: 764-767 (1972).

Table 39. Results of Parathion Application on Wheat Greenbug

Date	Application rate (lb AI/acre)	Yield increase (bu/acre)	Addition income at $1.67/bu ($/acre)	Application cost at AI $1/lb + $.50/application	Economic benefit ($/acre)	Source
1961	0.5	4.0	6.68	1.00	5.68	a/
	0.5	8.0	13.36	1.00	12.36	
	0.5	10.8	18.04	1.00	17.04	
	0.5	9.89	16.52	1.00	15.62	
1968	0.5	9.57	15.98	1.00	14.98	b/

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a/ Daniels, op. cit. (1962).
b/ Ward et al., op. cit (1972).

Peanuts

Efficacy Against Pest Infestation - Parathion is recommended for control of the southern corn rootworm on peanuts. Smith (1971)[1] evaluated several insecticides for control of the rootworm in Virginia in 1965, 1966 and 1967. In the 1965 tests, parathion at 2.0 lb/acre greatly reduced the amount of damaged fruit from the untreated checks in two experiments. The third experiment showed a higher rate of fruit damage than the check. In two tests in 1967, parathion-damaged fruit was reduced over 80% in one test but was about the same as the untreated check in the second experiment. Yield increases in the latter test were 1.1 lb/plot or 200 lb/acre. The author could not explain the lack of control by parathion since typical application tests have showed the adult rootworm to be highly susceptible to parathion.

Cost Effectiveness Of Pest Control - The price of peanuts averaged 14.5¢/lb in 1972 (Agricultural Statistics 1973); the cost of parathion was $1.00/lb (Chambers and Miller, 1974). The cost of application was $.50 per treatemtn. At these prices and costs the economic benefits for the above test amounted to a gain of $26.50/acre.

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[1] Smith, J. C., "Field Evaluation of Candidate Insecticides for Control of the Southern Corn Rootworm on Peanuts in Virginia," J. Econ. Entomol., 64:280-283 (1971).

Corn

Parathion is registered to control the European corn borer and Western corn rootworm, pests significantly affecting corn yields.

Efficacy Against Pest Infestation - Munson et al. (1970)[1] evaluated several insecticides for control of these pests in 1964 and 1965 at selected locations in Iowa. At Sanborn, Iowa in 1964, parathion at 1.0 lb/acre gave significant reductions in corn borer cavities and significantly lower net root ratings for corn rootworm control. Yields increased 113 and 97 bushels/acre over an untreated check for two plots with parathion applied at different dates. The lower yield increase occurred on the later date.

An experiment at Hamburg, Iowa in 1965 also showed significant reductions in net root ratings and corn borer cavities over the untreated. Yields, however, increased 10 and 5 bushels/acre over the untreated check for applications at different dates. The author concluded that one application of parathion can effectively control these pests.

Cost Effectiveness Of Pest Control - The price of corn averaged $1.20/bushel in 1972 (Agricultural Statistics 1973); the cost of parathion was $1/lb (Chambers and Miller,1974). The cost of application was $.50 per treatment. At these prices and costs the economic benefits ranged from $4.95 to $143.27/acre.

These results are summarized in Table 40.

Table 40. Results of Parathion Application on Corn Insect Pests

Date	Application rate (lb AI/acre)	Yield increase (bu/acre)	Additional income at $1.29/bu ($/acre)	Application cost at AI $1/lb + $.50 Application	Economic benefit ($/acre)	Source
1964	1.0	113	144.77	1.50	143.27	a/
	1.0	97	125.13	1.50	123.63	
1965	1.0	10	12.90	1.50	11.40	
1965	1.0	5	6.45	1.50	4.95	

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a/ Munson et al., op. cit. (1970).

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[1] Munson, R. E., T. A. Brindley, D. C. Peters, and W. G. Lovely, "Control of Both the European Corn Borer and Western Corn Rootworms with One Application of Insecticide," J. Econ. Entomol., 63:385-390 (1970).

Potatoes

Efficacy Against Wireworm Infestation - Parathion is recommended for control of the Pacific wireworm in the Northwest. Onsager (1969)[1] conducted experiments in 1965, 1966 and 1967 to evaluate insecticides for control of the wireworm in Eastern Washington. Parathion effectively controlled the wireworm as measured by a reduction in culls over an untreated check. Culls were reduced between 78 and 95% in the three tests. However, there appears to be no relation between yield and wireworm control since yields varied from a loss of 3,900 lb/acre to a gain of 4,400 lb/acre.

Cost Effectiveness of Wireworm Control - The price received for potatoes amounted to $2.55/cwt in 1972 (*Agricultural Statistics 1973*). The cost of parathion was $1/lb (Chambers and Miller, 1974). The cost of application was $.50 per treatment. At these prices and cost, the economic benefits vary from a loss of $103.45 to a gain of $108.70/acre from the use of parathion to control the wireworm.

These results are summarized in Table 41.

Table 41. Results of Parathion Application for Wireworm Control.

Date	Application rate (lb AI/acre)	Yield* increase (cwt/acre)	Additional income at* $2.55/cwt ($/acre)	Application cost at AI $1/lb + at $.50/application	Economic benefit* ($/acre)	Source
1965	3.5	(39)	(99.45)	4.00	(103.45)	a/
1966	4.0	2	5.10	4.50	9.60	
1967	3.0	44	112.20	3.50	108.70	

* Data in parentheses indicate decreases in yield, income and economic benefit.

a/ Onsager, *op. cit.* (1969).

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[1] Onsager, J. A., "Nonpersistent Insecticides for Control of Pacific Coast Wireworm," *J. Econ. Entomol.*, 62:1065-1067 (1969).

Potato Aphid Control - The results of a test reported in American Cyanamid Pesticide Petition 19[1] showed that a 15% parathion wettable powder applied at a rate of 1 lb/100 gal. (~0.15 lb/acre) in six applications to potatoes for control of the potato aphid in New York yielded an average of 400 bu/acre compared to an untreated check yield of 340 bu/acre. This is an increase of 60 bushel for 3,600 lb/acre which results in an economic benefit of $87.90/acre.

Leafhopper Control - In the same petition parathion (15% wettable powder) was tested for control of the potato leafhopper in Ohio in 1950. The insecticide was applied three times at a rate of 1 lb/100 gal. (~0.15 lb/acre) resulting in a yield of 733 bushels/acre compared to an untreated check which yielded 402 bushels. The application of 0.45 lb of parathion produced a 331 bushel (19,860 lb) yield increase resulting in an economic benefit of $504.48/acre.

Lima Beans - The *lygus hesperus* is a significant pest affecting yields of lima beans. Bushing et al. (1974)[2] evaluated insecticides for control of this pest at Davis, California, between 1969 and 1971 and concluded that parathion applied at about the period of first bloom increased yields significantly. The results of one test showed a yield increase of 2,305 lb/acre with the application of parathion. It had to be applied three times at 0.5 lb/acre because populations rebounded to new highs very quickly after each application.

The price of lima beans was $208.00/ton in 1972 *Agricultural Statistics 1973*; the costs of parathion was $1/lb (Chambers and Miller, 1974; the cost of application was $.50 per treatment. Using these prices and costs the additional income from this test which yielded 2,305 lb/acre would be $239.72/acre. Subtracting the cost of parathion of $236.22/acre.

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[1] American Cyanamid Company, Pesticide Petition No. 19, Malathion - Summary of Data, EPA Registration Files.

[2] Bushing, R. W., and V. E. Burton, "Partial Pest Management Programs on Dry Large Lima Beans in California: Regulation of *L. hesperus*," *J. Econ. Entomol.*, 67:259-261 (1974).

Peas

One test result was available from the EPA files regarding parathion use on peas to control the pea aphid. American Cyanamid Pesticide Petition 252[1] reported that the yield of peas when treated with 40 lb/acre of 1% parathion dust in a Maryland test in 1951 was 29.5 lb/plot. A comparable untreated check yielded 20.1 lb for an increase of 9.4 lb.

In 1972 peas sold for an average of $112/ton and yields were 1.36 tons/acre (Agricultural Statistics 1973). Using the yeild increase of 44.8% from the above test and the price of $112/ton this would result in yield increase of 0.61 tons or $68.42/acre. At a cost of parathion of $1/lb (Chambers and Miller, 1974) and application cost of $.50 per treatment, the resultant economic benefit would be $67.52/acre.

Strawberries

Schaefers et al. (1972)[2] evaluated parathion for control of the tarnished plant bug on strawberries and concluded that two applications of parathion at 0.5 lb/acre appeared to provide satisfactory reduction in injury, but the advantages of such a program was not evident. These results are summarized in Table 42.

Table 42. Results of Parathion Application on Strawberry/Tarnished Plant Bug.

Date	Application rate (lb AI/acre)	No.	Number of berries	Injury (%)	Increased yield (%)	Source
1971	0.5	2	8,065	24	31	a/
	0.5	1	8,019	34	21	
	check	--	8,652	55	--	
	0.5	1	2,721	35	36	
	check	--	2,132	71	--	

a/ Schaefers, op cit. (1972).

The prices received for strawberries in 1972 averaged $24/cwt and yields averaged 105 cwt/acre (Agricultural Statistics 1973). The yield increases from the above tests ranged from 21% to 36% over the untreated berries. At an income of $2,520 lb/acre of berries, the additional income.

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[1] American Cyanamid Company, Pesticide Petition No. 252, Malathion - Summary of Data, EPA Registration Files.

[2] Schaefers, G. A., "Insecticidal Evaluations for Reduction of Tarnished Plant Bug Injury in Strawberries," J. Econ. Entomol., 65:1156-1160. (1972).

from parathion use at the above yield increases would range from $529.20 to $907.20/acre. Subtracting the parathion costs of $1/lb and the application cost of $.50 per treatment, the economic benefit would range from $527.70 to $905.70 acre.

References

Adkisson, Perry L., and S. J. Nemec, "Comparative Effectiveness of Certain Insecticides for Killing Bollworms and Tobacco Budworms," Publication B-1048, Texas Agr. Exp. Sta. (1966).

Adkisson, Perry L., and S. J. Nemec, "Insecticides for Controlling the Bollworm, Tobacco Budworm, and Boll Weevil," MP-837, Texas Agr. Exp. Sta. (1967).

Agricultural Statistics, 1973, U.S. Department of Agriculture (1973).

American Cyanamid Company, Pesticide Petition No. 19, Malathion - Summary of Data, EPA Registration Files.

American Cyanamid Company, Pesticide Petition No. 252, Malathion - Summary of Data, EPA Registration Files.

Bost, W. M., Director, "Cooperative Extension Service Mississippi State University, Mississippi State Mississippi, Summary of Test Results at Stoneville and Verona, Mississippi, and Costs of Pesticides," personal letter to Mr. David F. Hahlen (1974).

Bushing, R. W., and V. E. Burton, "Partial Pest Management Programs on Dry Large Lima Beans in California: Regulation of *L. hesperus*," *J. Econ. Entomol*., 67:259-261 (1974).

Cate, J. R., Jr., and D. G. Bottrell, "Field Evaluation of Insecticide Treatments for Control of the Sorghum Midge," PR-2866, pp. 13-15, Research on Grain Sorghum Insects and Spider Mites in Texas, Texas Agr. Exp. Sta. (1971).

Cate, J. R., Jr., D. G. Bottrell, and G. L. Teetes, "Management of the Greenbug on Grain Sorghum. I. Testing Foliar Treatments of Insecticides Against Greenbugs and Corn Leaf Aphids," *J. Econ. Entomol*., 66:945-951 (1973).

Chambers, William, and Daniel Miller, Farmland Industries, Kansas City, Missouri, conversation (1974).

Daniels, N. E., "Insecticidal Greenbug Control in Grain Sorghum," PR-2868, pp. 16-20, Research on Grain Sorghum Insects and Spider Mites in Texas, Texas Agr. Exp. Sta. (1971).

Daniels, N. E., "Insecticidal Control of the Greenbug," Progress Report 2247, Texas Agr. Exp. Sta. (1962).

DePew, L. J., "Evaluation of Foliar and Soil Treatments for Greenbug Control on Sorghum," *J. Econ. Entomol*., 64:169-172 (1971).

Harvey, T. L., and H. L. Hackeratt, "Chemical Control of a Greenbug on Sorghum and Infestation Effects on Yields," *J. Econ. Entomol*., 63: 1536-1539 (1970).

Headley, J. C., and J. N. Lewis, *The Pesticide Problem: An Economic Approach to Public Policy*, Resources for the Future, Inc., pp. 39-40 (1967).

Huddleston, E. W., D. Ashdown, B. Maunder, C. R. Ward, G. Wilde, and C. E. Forehand, "Biology and Control of the Sorghum Midge. I. Chemical and Cultural Control Studies in West Texas," *J. Econ. Entomol*., 65:851-855 (1972).

Meisch, N. V., George L. Teetes, N. M. Randolph, and A. J. Bockholt, "Phytotoxic Effects of Insecticides on Six Varieties of Grain Sorghum," *J. Econ. Entomol*., 63:1516-1517 (1970).

Munson, R. E., T. A. Brindley, D. C. Peters, and W. G. Lovely, "Control of Both the European Corn Borer and Western Corn Rootworms With One Application of Insecticide," *J. Econ. Entomol*., 63:385-390 (1970).

Onsager, J. A., "Nonpersistent Insecticides for Control of Pacific Coast Wireworm," *J. Econ. Entomol*., 62:1065-1067 (1969).

Plapp, F. W., Jr., "Insect Resistance in *Heliothis*: Tolerance in Larvae of *H. virescens* as Compared With *H. zea* to Organophosphate Insecticides," *J. Econ. Entomol*., 64:999-10002 (1971).

Schaefers, G. A., "Insecticidal Evaluations for Reduction of Tarnished Plant Bug Injury in Strawberries," *J. Econ. Entomol*., 65:1156-1160 (1972).

Smith, J. C., "Field Evaluation of Candidate Insecticides for Control of The Southern Corn Rootworm on Peanuts in Virginia," *J. Econ. Entomol.*, 64:280-283 (1971).

U.S. Department of Agriculture, *Agricultural Statistics 1973*.

U.S. Environmental Protection Agency, *EPA Compendium of Registered Pesticides*, Vol. III, (1973).

U.S. Tariff Commission, *Synthetic Organic Chemicals*, U.S. Production and Sales, 1972, TC Publication 681 (1973).

Ward, C. R., E. W. Huddleston, D. Ashdown, J. C. Owens, and K. L. Polk, "Greenbug Control on Grain Sorghum and the Effects of Tested Insecticides on Other Insects," *J. Econ. Entomol.*, 63:1929-1934 (1970).

Ward, C. R., J. Owens, D. Ashdown, E. Huddleston, and W. Turner, "Greenbug Control on Wheat in 1967-1969," *J. Econ. Entomol.*, 65:764-767 (1972).

Wolfenbarger, D. A., "Tobacco Budworm: Cross Resistance to Insecticides in Resistant Strains and in a Susceptible Strain," *J. Econ. Entomol.*, 66:292-294 (1973).